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SOME CONSIDERATIONS CONCERNING THE ROENTGEN DIAGNOSIS OF PNEUMOCONIOSIS AND SILICOSIS*†

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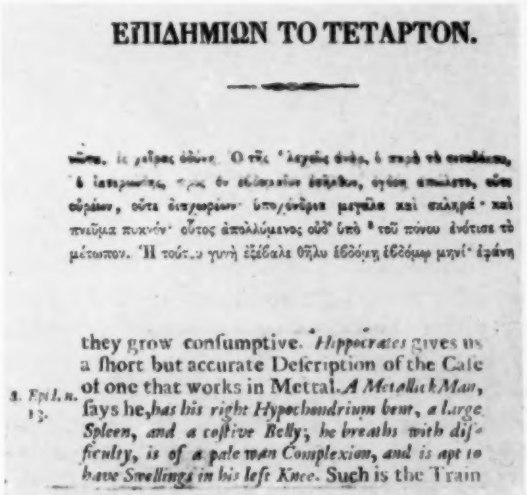
I AM grateful for the honor and conscious of the responsibility of speaking to you on this occasion, an evening dedicated to the memory of "Preston Manasseh Hickey." This is not the time for me to attempt to refresh your minds concerning Doctor Hickey's contributions to medicine. Many of these are a matter of record. Doctor Hickey was a physician, scientist and teacher. He possessed such qualities as understanding, honesty, reason and justice which were a source of inspiration and satisfaction to those of us who were privileged to know him.

The subject which I have chosen for presentation this evening is one of timely interest, although some of the symptoms and changes in the lungs produced by inhalation of dust in certain occupations were described many centuries ago (Fig. 1 and 2). It would be unwise, even were it possible, to present to you in an orderly manner the history of pneumoconiosis, but I have selected a few historical references to illustrate the antiquity of the disease and its distribution.

One of the earlier references concerned with the dangers of dust exposure is Pliny's[29] (23–79 A.D.) description of the devices used by refiners to prevent inhalation of the "fatal dust" (Fig. 3).

In the Renaissance, physicians and mining engineers were aware that the metal



ΕΠΙΔΗΜΙΩΝ ΤΟ ΤΕΤΑΡΤΟΝ.

they grow conſumptive. *Hippocrates* gives us a ſhort but accurate Deſcription of the Caſe of one that works in Mettal. *A Metallick Man*, ſays he, *has his right Hypochondrium bent, a large Spleen, and a coſtive Belly; he breaths with diſficulty, is of a pale wan Complexion, and is apt to have Swellings in his left Knee.* Such is the Train

3. Epid. 6. 13.

FIG. 1. An early description of the symptoms of a worker in metal.

* Hickey Memorial Lecture, delivered before Wayne County Medical Society, Monday evening, April 6, 1942.

† From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia.

miner suffered from shortness of breath and died prematurely. In 1556 Agricola[1] described the perils of mining and the pestilential air breathed by miners. Ramazzini[30] in 1700 called attention to the possible relationship between dust inhalation and consumption (Fig. 4). Anatomists had described "heaps of sand" in the lungs of stone cutters and they called the condition

In more recent years numerous studies and investigations have been carried out in many countries. It is only possible for me to mention a few. Some of the more important work concerned with the diagnosis, control and compensation for silicosis has originated in South Africa in connection with the gold mining industry.[16,21,33] In England, the reports of Haldane[14] definitely

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DISSERTATIO INAUGURALIS MEDICA,
DE
SPADONE
HIPPOCRATICO
LAPICIDARUM SEEBERGENSIUM HÆMOPTYSIN ET PHTHISIN PULMONALEM,
VULGO:
Der Seeberger Steinbrecher Krankheit
PRÆCEDENTE.
QUAM
DIVINA ANNUENTE GRATIA,
ET
GRATIOSÆ FACULTATIS MEDICÆ CONSENSU,
PRÆSIDE
DN. GEORGIO DAN. COSCHWITZ,
MED. DOCT. EJUSDEMQUE IN ALMA FRIDERICIANA PROF. PUBL. ORDIN. POTENTISS. REGIS BORUSS. IN TRACTU MANSFELDENS. ET APUD PALAT. HALENS. PHYSICO MERITISSIMO,
PATRONO, AC PROMOTORE OMNI OBSERVANTIÆ CULTU DEVENERANDO,
PRO GRADU DOCTORIS,
Summisque in arte Medica Honoribus, Insignibus ac Privilegiis
More Majorum solenni obtinendis,
Horis ante & pomeridianis Anno MDCCXXI. Die Jul.
Publico Eruditorum Examini submittit,
AUCTOR ET RESPONDENS
JOHANNES BUBBE, p. t. Med. Pract. in Præfect. Seeberg.
HALÆ MAGDEB. LITERIS CHRISTIANI HENCKELII, ACAD. TYP.

MEMBRUM II.
Quod definitionem [illegible] explanat.

ESsentia Spadonis, ut omnibus innotescat, necesse est ut post definitionem nominalem, recenseamus quoque realem. Ponimus ergo Spadonem definiri posse sequentibus:

Est tumor mollis, concolor & cedens internus a distensione vel ruptione arteriæ aut venæ, sanguine localiter velut in sinu congesto, endemice contingens iis operariis, qui in Lapicidiis Seebergensibus lapides effodiendo & aptando occupati sunt.

"Spadonism" we can define as follows: it is a soft tumor, varicolored, caused by internal distention or eruption of an artery or vein, likewise by a local blood congestion, as occurs in those workers in the stone quarries of Seeberg who are engaged in quarrying and polishing the stone".

FIG. 2. The title page and an appropriate reference from Spadone's dissertation on pulmonary phthisis in the Seeberg miners.

"phthisis." Thackrah [32] in 1831 noted that sandstone workers died before the age of forty, but there was no unusual incidence of lung diseases in brick and limestone workers. Although the effect of various dusts on the lungs had been recognized previously by other writers, Zenker[36] in 1867 is given credit for having coined the term pneumonokoniosis (Fig. 5). Shortly thereafter, in 1870, Visconti[35] described a pathological condition of the lungs resulting from inhalation of silica, which he called silicosis.

focused governmental attention on rock dust as being the causative agent of the lung injury, and pointed out that ordinary phthisis was different from that occurring in the Cornish tin miners. In 1915 Collis[3] expressed the theory that free crystalline silica was responsible for the lung damage and that it made the individual more susceptible to tuberculosis. In Canada, the investigative work at the Banting Institute has been noteworthy, especially that concerned with prevention. Denny *et al.*[4] have reported that animals dusted with quartz

and very small quantities of metallic aluminum showed very little if any fibrosis.

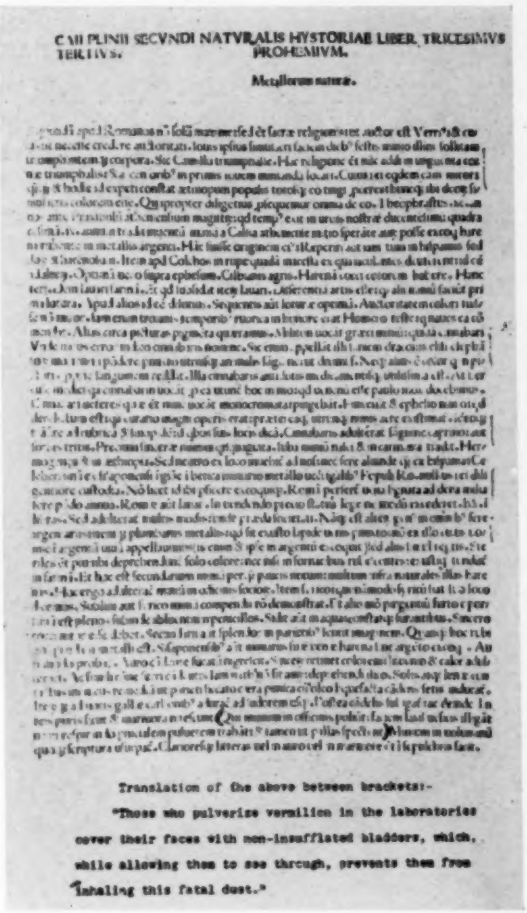
In the United States, the investigations carried on at the Saranac Lake Laboratory under the direction of Leroy U. Gardner have added tremendously to our knowledge of the development of pneumoconiosis and silicosis in the experimental animal. Likewise the work done by the Harvard School of Public Health on the engineering methods of dust determination and control, and the field studies of the United States Public Health Service and Metropolitan Life Insurance Company have done much to improve the atmospheric conditions in many industries.

Pneumoconiosis is a generic term used to describe all forms of pulmonary reactions to dust lodging in the lungs. These changes may or may not be demonstrable by the roentgen examination. Other terms such as siderosis and anthracosis are special names which serve to indicate the lung condition occurring in certain industries (iron and coal mining).

Silicosis is a form of pneumoconiosis which is thought to be due to the specific action upon the lung tissue of chemically free silicon dioxide in finely divided form and which produces changes that can be demonstrated in many instances on a roentgenogram. It is probable that all dusts, with the exception of chrome, free silica and certain silicates, produce very few changes in the lung that can be recognized with assurance on the roentgenogram.

In order to produce lung changes it is necessary for the silica dust to reach the lung alveoli, to get into the interstitial tissues either through the agency of macrophages (dust cells) or by way of alveolar pores, and to be deposited in sufficient concentration at some spot where either through its inherent physicochemical properties or together with some other factor or factors, as yet unknown, an abnormal amount of fibrous tissue formation may be stimulated.

From an examination of the mineral particles which have gained access to the lung it seems that the protective mechanisms of the respiratory tract exclude most of the particulate material greater than 10 microns in diameter. Likewise it is accepted generally that the smaller silica particles, 0.5 to 2.5 microns in size, are more likely to produce lung damage than are the larger particles. It is believed by some investigators that certain dusts (diluents or contaminating dusts) may have modifying effects upon the action of free silica in the lung tissues; for instance, carborundum and aluminum oxide may retard and alkaline soap powders may accelerate the usual reaction to free silica. There is some uncertainty about this hypothesis, however,



C. PLINII SECVNDI NATVRALIS HYSTORIAE LIBER TRICESIMVS TERTIVS.

PROHEMIVM.

Metallorum natura.

Translation of the above between brackets:-

"Those who pulverize vermilion in the laboratories cover their faces with non-insufflated bladders, which, while allowing them to see through, prevents them from inhaling this fatal dust."

Fig. 3. Pliny's reference to protective devices for those who pulverize vermilion.

as Gardner[9] says that "definite proof of accelerators to the action of silica is yet lacking; it has been suggested that cases of 'rapid silicosis' in human beings may be due to excessive exposures to silica or unusual fineness with or without associated infection." It is evident, therefore, that in order to evaluate a dust hazard one must have a knowledge of the behavior of the dust in the course of its production and while it is suspended in the air, and its behavior in the respiratory system, as well as an understanding of its specific action in the lung tissue.[15] *The knowledge that a workman is exposed to dust released in the processing of a material containing free silicon dioxide is insufficient evidence in itself to justify one in stating that the atmosphere at the breathing level contains harmful quantities of free silica dust.*

The reasons for this observation are supported by ventilation and sanitary engineers familiar with many of the differences in the behavior of dusts and some of the factors concerned may be abstracted as follows:[15]

1. The composition of the air borne dust may differ from that of the parent material.
2. The composition of the dust retained in the deeper portions of the lung may differ from that of the air borne dust.
3. The amount of dust retained in the respiratory system and the relative amounts retained in the upper portion of the tract and that found in the alveoli vary with different dusts.

A Treatise of the Diseases of Tradesmen, Shewing the various Influence of particular Trades upon the State of Health; With the best Methods to avoid or correct it, and useful Hints proper to be minded in regulating the Cure of all Diseases incident to Tradesmen.

Written in Latin by Bern. Ramazzini, Professor of Physick at *Padua*.

And now done in English.

LONDON, Printed for *Andrew Bell*, *Ralph Smith*, *Daniel Midwinter*, *Will. Hawes*, *Will. Davis*, *Geo. Strahan*, *Bern. Lintot*, *Ja. Round*, and *Jeff. Wale*. 1705.

of Tradesmen. 163

CHAP. XXIV.

Of the Diseases of Stone-cutters.

WE come next to the Diseases of Stone-hewers, Statuaries, Stone-cutters, and that sort of Workmen; for in hewing Marble or Stones out of the Rock, in polishing and cutting them, they oftentimes suck in, by inspiration, the sharp, rough, and corner'd small Splinters or Particles that flie off; so that they are usually troubled with a Cough, and some of 'em turn Asthmatick and Consumptive. Add to this the metallick Vapour exhaling from the Marble Stones, *&c.* which manifestly affects the Nostrils, and the Brain. Thus the Stone-cutters that work upon the *lapis Lydius*, are said to be so affected both in the Head and the Stomach, by the heavy Smell that constantly exhales from thence, that sometimes they are forced to Vomit: And in dissecting the Corps of such Artificers, the Lungs have been found stuffed with little Stones. *Diemerbroek* gives a curious Relation of several Stone-cutters that dy'd Asthmatick, and were open'd by him; in whose Lungs he found such heaps of Sand, that in running the Knife thro' the Pulmonary Vesicles, he thought he was cutting some Sandy Body. He adds, that he was inform'd by a Master Stone-cutter, that in cutting Stones there rises such a subtile Dust, as is able to penetrate thro' Ox Bladders hung in the Shop, insomuch that in the space of one Year he found a handful of that Dust in the Cavity of the Bladder: And this very Dust he took to be the cause of the Death of many unwary Workmen. *L. 2. Anat. c. 13.*

Our

FIG. 4. Title page and reference from the English translation of one of Ramazzini's works.

4. The particle size of the alveoli dust differs from that of the air borne material.

5. The flocculating properties of dust vary; this affects the retention in the respiratory tract, its penetration to the alveoli, and the behavior of phagocytes toward the dust.

6. The rate of phagocytosis varies from one dust to another.

7. The toxicity of silica is apparently reduced in the presence of certain other materials.

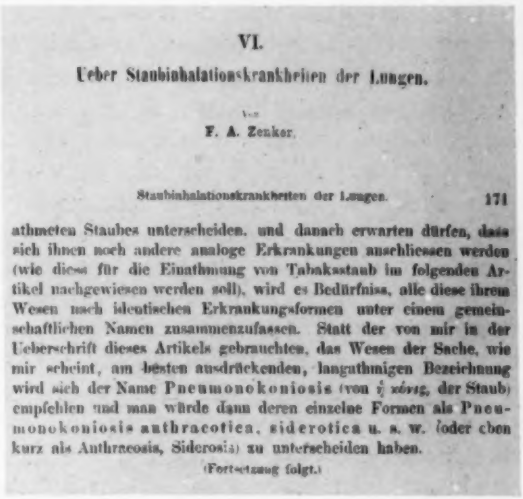
According to Hatch[15] the relative significance of these several aspects of dust behavior is not fully understood but their importance has been demonstrated both in the laboratory and in industrial experience.

THE PROTECTIVE INFLUENCE OF THE RESPIRATORY SYSTEM

The respiratory system is so constituted that it offers a definite protection against dust laden atmosphere entering the alveolar structure of the lungs. The peculiar construction of the nasal cavities, the hairs in the nares, the moist turbinated bones, and the ciliary action of the tracheobronchial mucosa are factors in preventing considerable amounts of dust (especially the larger particles) reaching the alveoli. The amount which does penetrate to the lung depths varies somewhat with the frequency and depth of breathing.[15] The air velocity is highest in the trachea and the restricted passages of the nasal fossae and gradually decreases as the alveoli are approached. According to Hatch, the air flow in the bronchi is approximately one-half of the tracheal velocity and in the bronchioles it is further reduced to one one-thousandth. At the entrance of the alveoli the rate of motion is only 1/100,000 of the tracheal velocity. Within the alveoli, the air movement relative to the alveolar walls approaches zero. Another important consideration is that the air flow moves in different directions with the phase of respiration and therefore varies from zero to a maximum in every cycle. Likewise it is possible that not all of the inspired air reaches the alveoli.

It would seem reasonable to believe, therefore, that several factors concerning dust exposure and certain physiological processes may become operative in a given individual.

a. The amount of dust that reaches the lung alveoli varies with the frequency and depth of breathing.



VI.

Ueber Staubinhalationskrankheiten der Lungen.

von

F. A. Zenker.

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athmeten Staubes unterscheiden, und danach erwarten dürfen, dass sich ihnen noch andere analoge Erkrankungen anschliessen werden (wie diess für die Einathmung von Tabaksstaub im folgenden Artikel nachgewiesen werden soll), wird es Bedürfniss, alle diese ihrem Wesen nach identischen Erkrankungsformen unter einem gemeinschaftlichen Namen zusammenzufassen. Statt der von mir in der Ueberschrift dieses Artikels gebrauchten, das Wesen der Sache, wie mir scheint, am besten ausdrückenden, langathmigen Bezeichnung wird sich der Name **Pneumonokoniosis** (von ἡ κόνις, der Staub) empfehlen und man würde dann deren einzelne Formen als **Pneumonokoniosis anthracotica, siderotica** u. s. w. (oder eben kurz als Anthracosis, Siderosis) zu unterscheiden haben.

(Fortsetzung folgt.)

FIG. 5. An illustration of a portion of Zenker's article in which the name "pneumonokoniosis" was coined.

b. The dust collecting efficiency of the upper respiratory tract may vary considerably depending on the condition of the nasal fossae and its included structures and whether an individual is a mouth breather or not.

c. The dust reaching the alveoli has a particle size approximately 1 micron or less in size because of the regional selective efficiency of the respiratory tract in relation to size, and therefore the dust reaching the alveoli may have a different composition from that which was breathed in.

Hatch[15] states that the dust collecting efficiency of the respiratory system has an average value of 50 per cent for industrial dusts. In a factory with a dust concentration of 5 mg. per cubic meter, an individual breathing 10 cubic meters per day will re-

tain over 5 gm. of dust per year, or 150 gm. in a working span of thirty years. Because of the anatomical structures concerned with the removal of the foreign material, only a small portion of that amount is generally retained. Thus silicotic lungs may contain no more than 6 gm., which is only a small percentage of the original dust inhaled.

What happens to the dust that lodges in the air sacs? Some of it may remain in the alveoli. Some gains entrance into the interstitial structures, through the agency of macrophages (dust phagocytes) or alveolar pores.[19] A large amount may pass into the lymphatics to be transported along the lymphatic channels to adjacent lymphoid deposits or lymph nodes. Some of the dust may get into the general circulation by way of the tracheobronchial or hilar lymph nodes. Some of the dust laden phagocytes are deposited along the lymphatic channels located subpleurally and those accompanying the vascular structures and bronchial tree.

The functions of the lymphatic system of the lungs are not known, but at least one is thought by some writers to be concerned with removing foreign material from the lung. Miller[24] describes superficial and deep sets of lymphatics, the former coursing through the pleura and the latter along the sheaths of the blood vessels and bronchi. The lymph flow of these two sets for the most part is directed towards the hila. They intercommunicate and if for some reason the deep set becomes obstructed, the lymph flow may then reach the hilum by way of the superficial lymphatics. Some of the lymphatics from the lower portion of both lungs pass through the domes of the diaphragm to reach the preaortic lymph nodes.

As stated above, one is not certain as to the functions of the lymphatics, but it is believed that the powerful movement of the domes of the diaphragm, the movements of the ribs through the agency of the intercostals and other accessory muscles, the lobation and the respiratory movements of the lung exert a profound influence on the lymphatic flow.[17,20] In so doing, the respiratory movements and massage action of the ribs assist not only in the transportation of material within the lumen of lymphatic channels, but may exert some influence on dust particles deposited outside of those structures. In favor of such a hypothesis, it has been shown that pneumothorax diminishes lymph flow.[5] Likewise, wherever there is fixation or limitation of movement of the lungs, one finds an increased deposition of pigment or dust particles along the pleural surfaces of the lung. It is common knowledge that more pigmentation of the lung from dust occurs in the region of the intercostal spaces than at the site of the ribs, except in those individuals where the pigmentation is extreme.

PATHOLOGICAL CONSIDERATIONS

In this presentation, I am only concerned with the benign nonspecific pneumoconiosis and silicosis.

It is in the phagocyte that dust first exerts its specific influences on living cells and that differences due to physicochemical composition of the irritant become manifest.[10] Inert substances like aluminum oxide have no effect on the structure of the cell, although they may retard or stop its locomotion by their mass within its cytoplasm.[10] It is presumed that if the dust gets into the interstitial tissues without the agency of the phagocyte, the same conditions are operative.

Toxic free silica quickly injures the phagocytes, and its effect is directly proportional to the number and size of the ingested particles.[9] The migratory phagocytes concentrate silica in and about the lymphatic system of the lungs. The ultimate result is a nodule composed of connective tissue whose fibers are modified characteristically by the silica. Ultimately these parenchymatous nodules may attain a diameter of 3–4 mm.[10] The degenerative change produced by free silica within the cell closely simulates those in the epithe-

lioid cells of tuberculosis.[11] A reason for the similarity of the changes produced by silica and tuberculosis has been offered by Fallon.[8] It is his belief that the irritation produced by silica and tuberculosis liberates toxic phospholipids which are responsible for the proliferation that results in the granulomatous nodule.

ROENTGEN CONSIDERATIONS

It is generally conceded that the roentgenographic examination, properly done, is the most precise method at our command for demonstrating visible shadows of pathological changes produced by pneumoconiosis or silicosis in the living individual. There are, however, many other conditions that produce shadows in the roentgenogram which may simulate some of the various shadows found in pneumoconiosis.[28] In order to evaluate correctly the various shadows observed in a roentgen study of the chest, it is necessary for one to possess some knowledge and experience concerning such an examination, and certain information about the patient being studied. Some of the more important requirements may be enumerated as follows:

1. A knowledge of the anatomy of the chest and some of the physiological manifestations of the various structures contained therein; an understanding of the histology of the lungs and of their lymphatic system.

2. A thorough familiarity with roentgenoscopic and roentgenographic appearances of the normal structures of the chest and their permissible variations.

3. A clear perception of the pathology of pneumoconiosis and of lesions that give a somewhat similar roentgenographic appearance.

4. Some knowledge of the history of the individual, especially the occupational record and familiarity with the physical signs in the particular patient.

5. Some information concerning the industrial process that is responsible for the production of the dust. Dust counts at breathing levels and chemical analyses of the dust are exceedingly important when available.

6. A comprehensive roentgen examination.

If all of the above information is available, one should be able to render a diagnosis which although presumptive is likely to be correct in the majority of instances. Very often, however, one gives an opinion on insufficient data and in so doing referring physicians become confused and injustices occur.

I have always regarded the roentgen examination as a consultation, and used as such it is likely to be more valuable. An ideal program for the diagnosis of pneumoconiosis in the living would include a study by a group, the members of which would be a general physician, a rhinologist, a bronchologist, a clinical pathologist, a specialist in tuberculosis, an engineer interested in industrial hygiene and a general roentgenologist. Such a program is too ambitious for practical purposes and in the majority of instances the referring physician tells the individual that he has pneumoconiosis or silicosis on the basis of a history of dust exposure, a physical examination which is thought to exclude other conditions that might produce similar symptoms and the presence of abnormal shadows in roentgenograms of the chest which are compatible with those found in pneumoconiosis. The fact that diagnoses are made in this manner places a tremendous responsibility on the roentgenologist, for he must not only describe the roentgen appearances, but after he has done so he must correlate the other information which has been supplied to him with the roentgen observations, and arrive at a tentative or presumptive diagnosis. This is a safe procedure if we as radiologists fulfill our obligations to the referring physician and patient.

ROENTGEN APPEARANCES IN BENIGN NONSPECIFIC PNEUMOCONIOSIS

Gardner[10] includes under this classification "every known reaction to mineral dust except that due to the soluble poisons such

as lead, the specific diseases, silicosis and asbestosis, and 'roentgenologic conditions' such as *baritosis* and the *pseudo nodulation* of *arc welders*."

Experimental investigations have shown that most nonsiliceous dusts exert no appreciable effects on connective tissues, the reaction is not progressive and does not terminate in a fibrosis appreciable on gross examination.[19] *It would seem, therefore, that if abnormal shadows are observed in roentgenograms of individuals exposed only to nonsiliceous dusts some other condition is present.*

Many physicians interested in pneumoconiosis and whose opinions are respected make a diagnosis of such a condition because of the observation of what they call "an exaggeration of the lung markings," and oftentimes the interpretation is based on a study of a single film. Are we justified in making a presumptive diagnosis on such evidence? It would seem that if Gardner's statements[10] concerning the absence of fibrosis appreciable on gross examination are true and the thoroughly known limitations[27] of the roentgenographic study of the chest are observed, we should *examine critically positive reports* of pneumoconiosis based on suggestive clinical and occupational data and roentgen evidence of "so-called" prominence of the lung markings.

We, as physicians and as roentgenologists, have been anxious to improve our diagnostic ability in pneumoconiosis through the agency of chest studies of individuals exposed to dust. We have been told by the experimental investigators what happens in animals under controlled circumstances, and by the pathologists what has happened in the postmortem lungs of humans. We also know from experience that when collective and serial roentgenographic studies are available on a patient whose lungs at death show evidence of pneumoconiosis one is likely to *magnify in terms of disease slight variations* (without nodules) *from the normal pattern* in the early examinations.

Some years ago the late Dr. Pancoast and I proposed a classification of "*perivascular, peribronchial, lymph node predominance*" to include the pathological changes under discussion in the roentgen diagnosis. After a few years we found that our opinion in regard to such an interpretation varied so much from day to day on the same films the classification was abandoned in our roentgen interpretation.

The time has arrived for us to take stock of our knowledge and abilities. There is still considerable work to be done on the manner in which the various pneumoconioses are produced. There are still too few postmortem studies in humans (that are free from the modifying influences of infection) in which good roentgenographic studies, dust counts and a careful occupational record are available for a correlated comparison. The roentgen examination, as made under conventional circumstances, has so many variables one must recognize that certain differences in appearances between two or more examinations cannot be regarded as significant without reservation. Another factor that is overlooked frequently is the difference in appearances between two chests. What is normal for one may be abnormal for another. The lung markings and often the dynamics are as variable as the features on different faces.

Does the presence of a scar or a lesion that is not completely healed produce roentgenographic evidence of pneumoconiosis? I do not know. Gardner[10] states that such lesions may retain abnormal quantities of dust particles and that such deposits may maintain a chronic inflammatory reaction, but he does not state whether such a combination might be demonstrable on a roentgenogram. Apparently there is no evidence to show that dusts, other than free silica, alter the natural susceptibility to tuberculosis.

Does pneumoconiosis from inert minerals produce disability? The lung is elastic and the alveolar walls are generally not involved even in extreme cases presenting diffuse pigmentation.[10] It therefore seems improbable that disability from pulmonary symptoms would occur under such circumstances. *It should be borne in mind that one cannot from a study of the chest by roentgen methods*

give any reliable opinion as to the extent of disability. Physicians interested in disability have developed elaborate methods for its detection and measurement, and in my opinion they should be the ones from whom we should seek such information.

SUMMARY

On the basis of the pathological findings and the known limitations of the roentgen examination *it would seem that in those instances where individuals are exposed to inert mineral dusts, one should not expect to be able to demonstrate conclusively changes that should be interpreted as roentgen evidence of pneumoconiosis.*

There will be instances in which a careful history fails to show any exposure to silica-containing dust and yet abnormal shadows will be seen in the roentgenogram. In such cases one is at a loss to know what to call the condition. My present plan, when absolute data are lacking other than the occupational record and physical findings, is to make a diagnosis of "*modified silicosis*" (Fig. 6*A* and *B*). This position is supported, especially when the individual is disabled to some extent, by Gardner[10] who states that "an apparently comprehensive occupational history and the histologic evidence of a single autopsy specimen does not warrant the inference that a mineral other than free silica has caused fibrosis."

There are two conditions due to certain dust exposure which can be diagnosed or suspected because of their roentgenographic appearances. Unless one is familiar with the dense, pseudonodulation occurring in baritosis and the less dense pseudonodulation found on at least one occasion in an arc

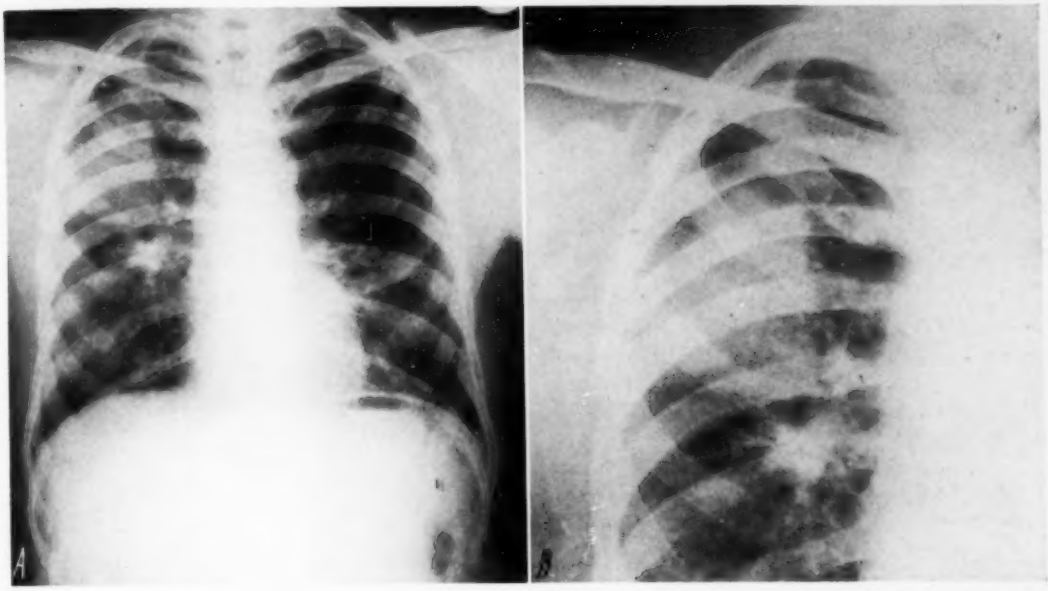


FIG. 6. *A. Anthracosilicosis.* E. C., male, aged forty-seven. This patient came to us with a history of having been exposed to finely divided coal dust. This coal dust is extremely fine and is used to make illuminating gas. Before taking up this work in 1916 this individual was a miner for seven years. According to a history he stated that he had had no silica dust exposure but the roentgen examination of the chest showed a massive lesion in the right upper lobe with a few small nodules distributed in both lower lung fields. From pathological investigation and animal experimentation one would not expect finely divided coal dust to produce such shadows. We therefore have made a diagnosis of anthracosilicosis on the basis of his having been exposed to silica when he was a coal miner. It is entirely possible that the eighteen years of exposure to finely divided coal dust, in his present occupation, may have contributed to the lesion in the right upper lobe as a result of a pre-existing scar in that region. *B*, the massive lesion in the right upper lobe is shown to better advantage.

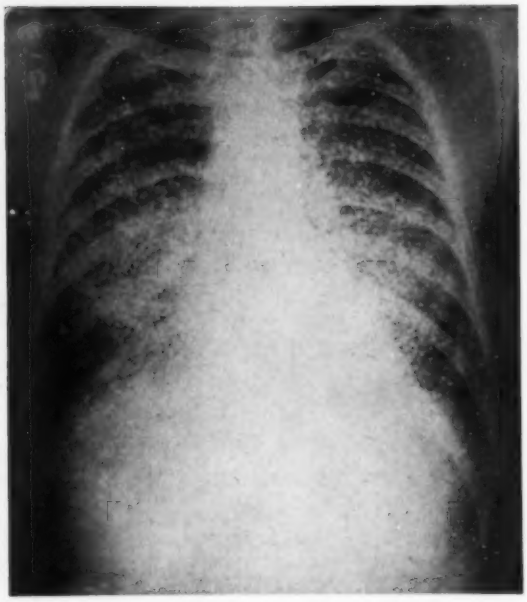


FIG. 7. *Baritosis.* This individual had no respiratory symptoms whatever. The condition was found in a routine investigation of workers in baryta ore. Compare the shadow densities with those in Figure 8. These densities are greater than those usually produced by silica nodulation.

welder, their presence may not be suspected.

Baritosis has been described by Arrigoni.[2] This form of pneumoconiosis has been found in the baryta mining industry of Italy and in a few workers in a Pennsylvania plant. The lungs are studded with small sharply circumscribed nodules (Fig. 7 and 8). In the experimental animal, the nodules are due to the collections of mineral dust and do not show evidence of fibrous tissue overgrowth. Individuals with baritosis do not have an increased susceptibility to tuberculosis.

The pseudonodulation occurring in an arc welder reported by Enzer and Sander[7] was due to the collections of iron particles. No fibrous tissue reactions were observed. Those who have observed large numbers of roentgenograms made of men who have done welding for years believe that the pseudonodulation occurs infrequently but when it does occur it may simulate shadows of nodulation occurring in silicosis.

ROENTGEN APPEARANCES IN SILICOSIS

The roentgen appearances of silicosis with and without infection and modified silicosis have been classified variously depending largely upon the recommendations of the investigators in certain countries.[28] Every exponent of a certain classification thinks his is the best for his particular needs. Classification of various stages of the disease will continue to be important until some better method is utilized to regulate compensation.

Pneumoconiosis implies a condition of fibrosis, occurring, so far as we know, in only one general way, but with certain variations dependent upon uncertain or unknown influences. It should be noted, however, that those who study a large number of occupations and industries find it difficult to adhere to a general division of

FIG. 8. *Probable silicosis with nodular predominance.* This patient's occupation was that of a laborer, for eighteen years, exposed to dust and irritating gases. Prior to that time he was a sand blaster for two years. He had no symptoms and no disability when subjected to the usual clinical tests. One might think that he has considerable disability if one depended on the roentgen manifestations. Compare the roentgen appearance of this case with that produced by baritosis in Figure 7. The shadows are quite similar.

stages, without making exceptions or creating certain variants. This becomes more complicated with each new occupation.

The clinical phenomena, of necessity, are due to certain pathological changes. Likewise, the roentgenographic appearances portray only those pathological changes which are macroscopic. The microscopic changes which cannot be seen roentgenologically may, however, be most important in producing disability. Everyone has seen instances in which extensive roentgen evidence of silicosis is accompanied by very little disability, whereas occasionally marked disability presumably due to silicosis is observed in individuals who have very little roentgen evidence of the disease (Fig. 8 and 9). Therefore, it would seem wiser to substitute, for a numerical progression of pathological changes typical for a few of the occupations, some other classification which need not place too much emphasis on the roentgen observations except in those instances in which there is good evidence of an active infection complicating the silicosis.

Very often one sees a roentgen diagnosis of "second stage silicosis." By that the radiologist infers that he has observed certain changes in the lungs which have been described as occurring in that stage. He does not mean necessarily that the individual has a progressive disease which in a few years will become extensive enough to be classified as third stage. There are occasions when silicotic lesions do progress if harmful exposures to silica dust are continued or some other factor, such as a superimposed infection, becomes operative. In other instances the lesion may remain unchanged for years, and the shadows of some of the lesions actually may get smaller.

The important thing to bear in mind concerning the roentgen report classifying silicosis into stages, is that *it should not in any way prejudice the clinician as to the presence, absence or degree of disability.*

Most of the changes that are seen in roentgenograms are those produced by silica and other dusts such as coal dusts.

Fig. 9. *Silicosis with nodular predominance.* N. N., male, aged forty-seven. Patient has been a soft coal miner for thirty years and is practically totally disabled because of dyspnea. The roentgen examination shows slight nodule formations distributed throughout both lungs. The nodules are not very large and not very diffuse. The above illustration gives one an idea as to the frequency of the nodules. There is a moderate amount of emphysema. There was a marked disability in this individual out of all proportion to the roentgen manifestations in the lungs. This is a good illustration of why it is unwise to attempt to evaluate disability from roentgen appearances.

Only a few opportunities have been available for roentgenologists to study men exposed in atmospheres of almost pure silica dust. Thus from a practical standpoint, all of the roentgen observations may be regarded as those of modified silicosis, and in this paper will be classified as simple silicosis and silicosis with infection.[25] An example of modified silicosis is anthracosilicosis, occurring in some of the hard coal miners.

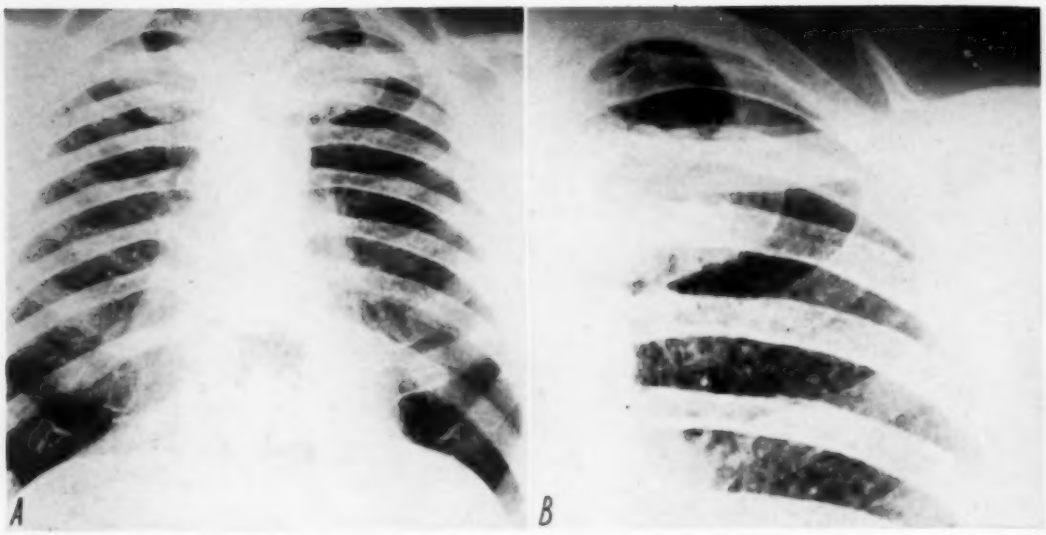
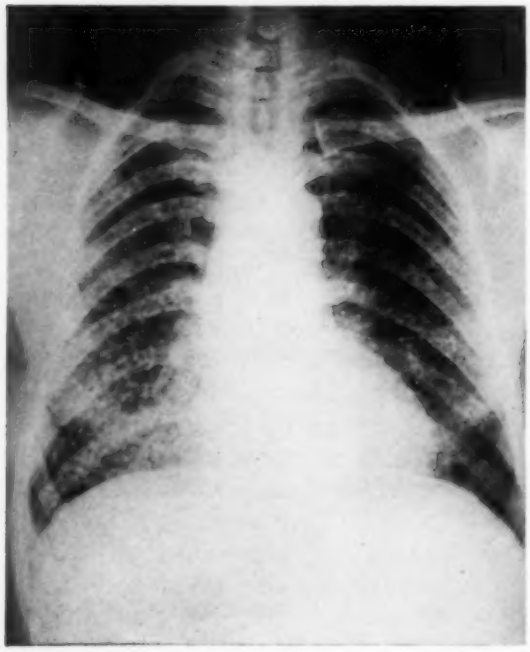


FIG. 10. *A. Very early nodular manifestations of silicosis.* C. C., male, aged forty. At the age of seventeen years he began to load coal at the head of the mine. At the age of thirty to forty he delivered coal by truck. At the age of forty he handled a tender. The roentgen manifestations are quite early. Note that the hila and trunk shadows are not increased in prominence and likewise the nodular shadows are not perfectly round but are varying shapes. This man had no disability. *B*, close-up of the left upper lobe.

SIMPLE SILICOSIS

The characteristic lesion in silicosis is a circumscribed nodule of hyaline fibrosis. The earliest lesions are invisible or are recognizable only microscopically or with a magnifying glass.[9] They are deposited along or within the lymph channels where they may impede lymphatic flow.[13,34] Gardner[10] states that when the lymph flow is retarded the phagocytes do not enter the lymphatics but collect here and there over the walls of the air sacs. Parenchymatous nodules may then develop which ultimately reach a size of 3–4 mm. in diameter. Pathologically, the nodules have well defined borders except when there are accumulations of non-siliceous dusts.



Bronchial tree, lymphatic and *vascular channels with supporting structures:* These structures are commonly referred to as trunk shadows and linear markings. Slight to moderate variations in these shadows are difficult to evaluate. Theoretically the

FIG. 11. *Anthracosilicosis with nodular predominance* occurring in a hard coal miner. W. J., male, aged forty-four. This man has worked in hard coal, cutting both rock and coal for a period of seventeen years. He worked regularly and felt perfectly well except for slight dyspnea. The roentgen examination shows a nodular predominance in an anthracosilicosis. While nodular predominance occurs in some individuals and not in others, the fact is difficult to explain.

shadows should be increased in prominence, but the chances for error in interpretation are increased greatly due to technical difficulties of obtaining identical serial roentgenograms and the personal equation in interpretation of the same roentgenograms on different days.

Lung changes: The earliest roentgen lesion that I accept as evidence of simple silicosis is the small, discrete, multiple shadow, 2–6 mm. in diameter, which is more or less uniform in size and density and does not disappear in a roentgenogram made with slight rotation (Fig. 10*A* and *B*, and 11) *Shadows that disappear with slight rotation are likely to be vascular.* The blood vessel shadows are denser and their borders are more sharply defined than are those of nodules. The shadows of the silicotic nodules are usually distributed along the vascular channels and the bronchial tree of both lungs, and at times they may be limited

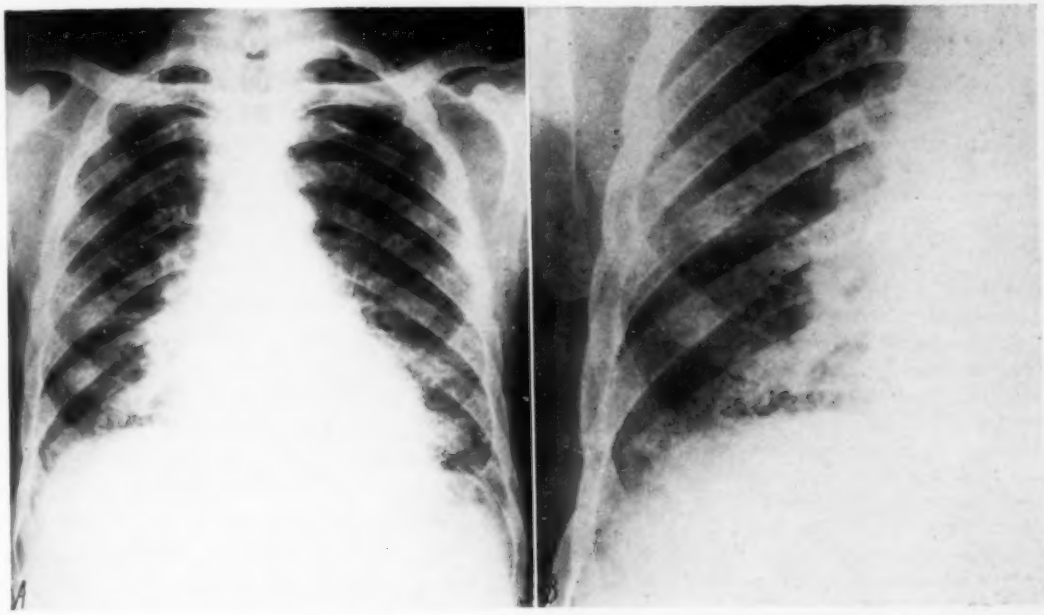


Fig. 12. *A. An oil pneumonia.* M. P., male, aged sixty. Referred with a clinical diagnosis of silicosis. This man was employed in a brass shop and worked in a large room in which there was good ventilation, high ceilings and tremendous floor space. He did polishing and cutting of copper and brass. In cutting the metal he used a lubricant which was a machine oil and, at the time the cutting was done, a smoke was created. The patient thought that he was suffering from silicosis because when some of the large castings would drop nearby, there was considerable oil around and although he could see dust from time to time it was really not a very dusty atmosphere.

The patient had paroxysms of coughing and at times would bring up considerable sputum, about a half cup at a time. He lost considerable weight, from 180 lb. to 136½ lb., and at the time I saw him he had marked disability. The man was very intelligent and helped considerably in arriving at the diagnosis. Films were made of the chest and one could see a fine diffuse mottling throughout the lower half of both lungs. The shadows were not those usually seen in silicosis but looked more like those of an infection. When I first saw the roentgenograms I never felt that the condition was a lipoid pneumonia for I would expect to find a little more consolidation in such a process, but, after questioning the patient more closely, the correct diagnosis was suspected and he was persuaded to have a bronchoscopic examination to see whether or not we could demonstrate evidence of oil droplets. Much to our amazement and surprise, we were not only able to demonstrate the oil droplets in the secretions removed from bronchoscopic aspiration, but to demonstrate oil droplets within phagocytes. In view of this observation we thought that we were dealing with an oil pneumonia rather than a silicotic process. One can readily see, therefore, that a diagnosis of silicosis could not have been made unless an adequate history had been obtained and the proper examination made to identify the cause of the lung shadows.

B, close-up of the right lower lung field.

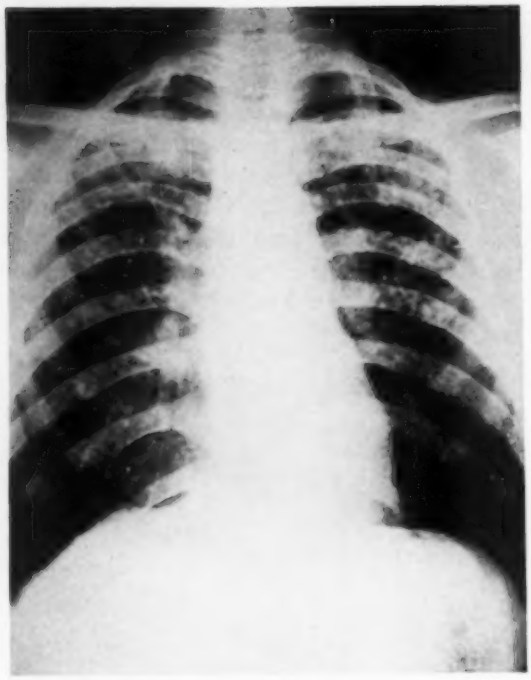


Fig. 13. *Uncertain.* Possibly tuberculosis or some other condition, the nature of which is uncertain. The illustration is included to show conditions from which silicosis must be differentiated. The patient was a male, aged thirty-seven, who came into the hospital with a history of dysentery and the chest examination was done as a part of the routine study. The patient had no cough and there was no expectoration and no dyspnea. The roentgen findings are those of nodulation and conglomerate shadows. A careful history was taken to obtain a history of dust exposure. The patient never had an abnormal dust exposure. He worked in Maryland for a while, as a farmer, and then became a stevedore. The cause of the shadows in the lung is still uncertain.

largely to one lobe. Not infrequently, even though a bilateral distribution of the shadows occurs, one does not see them in the apical, peripheral and lower portions of the lung fields.

The roentgen appearance of the shadows of the silicotic nodules are not as characteristic as one would wish. Some are round, some oval, some irregular, some large and some small. Most of the lesions have a uniform density, but some have shadow densities in their centers simulating that produced by calcium. The shadows of these lesions are called *nodulation*, which unwittingly implies that they can be distinguished from mottling due to tuberculosis or some other infectious process. I frequently have found that the periphery of the nodular shadows (silicotic) is so ill defined that in itself it cannot be differentiated from mottling in miliary and other types of tuberculosis and small shadows due to a generalized metastatic malignancy. In most instances, however, a correct diagnosis can usually be arrived at by correlating the roentgen findings with the history and clinical data (Fig. 12*A* and *B*, and 13).

One of the interesting questions that arises conderning the silicotic individuals as seen by the roentgenologist is that not all of them develop generalized nodulation that can be seen on the roentgenogram. This apparently is true even when individuals are working under similar conditions in the same industry, and up to the present time, no adequate explanation has been made.

When nodulation does occur, the roentgenologist finds it almost impossible to demonstrate a slowly progressive increase in size of the nodular shadows in serial films made at yearly intervals. In the few patients that I have had occasion to study under such circumstances, the roentgenograms of a given individual would be regarded as negative for two years, then the following year the shadows of nodules would become visible, and roentgenograms made in the succeeding year did not show any further increase in the size of the shadows. I have always been interested in whether nodulation is more likely to develop in the younger or the older individual. This question is likely to be answered as more experience with serial studies is obtained. My observations in one industry (almost pure silica) over a period of seven years tends to show that nodulation does occur in the young individual. The size and extent of the nodulation may not change for several years (limit of observation). These nodules have been observed in individuals who have had a silica dust ex-

posure of five to ten years. One wonders whether the nodulation seen in some of the older individuals who have worked for longer periods may likewise have developed during the five to ten years' exposure period and in certain instances failed to progress.

Generalized nodulation is often present with very little, if any, clinical evidence of disability (Fig. 8). Such an observation is extremely important to keep in mind for it will help to prevent such individuals from being thrown out of work and placed on compensation. Experience with such individuals has shown that if they are allowed to continue to work in a healthy atmosphere, the lesion may not progress, no unusual disability develops, and a family catastrophe or hardship due to loss of adequate compensation and unhappiness is prevented.

According to Gardner,[10] the nodular stage of silicosis is associated with a definite increase in susceptibility to tuberculosis. The infection may develop as a result of reactivation of a pre-existing, latent, tuberculous focus or as is much more likely, a new infection may become superimposed. Pre-employment and annual chest surveys in all industries will undoubtedly disclose many of the early tuberculous lesions that are not discovered by other means. If such individuals are removed from industries in which harmful amounts of silica are elaborated, not only the tuberculous individual but his fellow worker will be protected.

Another lesion generally included under the classification of simple silicosis is the small conglomerate lesion. Such lesions may result from a combination or coalescence of discrete nodules or the lesion may occur as such primarily.

Conglomerate lesions are usually localized and do not occur in the same portion of the lung in every individual. On the other hand, roentgenographic studies of individuals with such lesions more frequently show them in the upper half of the lung fields. Microsopic examination[10] of the tissues from such areas oftentimes reveals no evidence of infection. The nodules seem to be closer together than in other portions of the lung; they are less uniform in size, and they are embedded in a matrix of diffuse, fibrous tissue having the same characteristic appearance as that forming the nodules themselves. It is possible that the conglomeration may have occurred because the portion of the lung in question was previously damaged by a localized, inflammatory process occurring before or during the early period of the dust exposure.[10] More dust would tend to accumulate in such an area and possibly the nodules would develop irregularly and closer together.

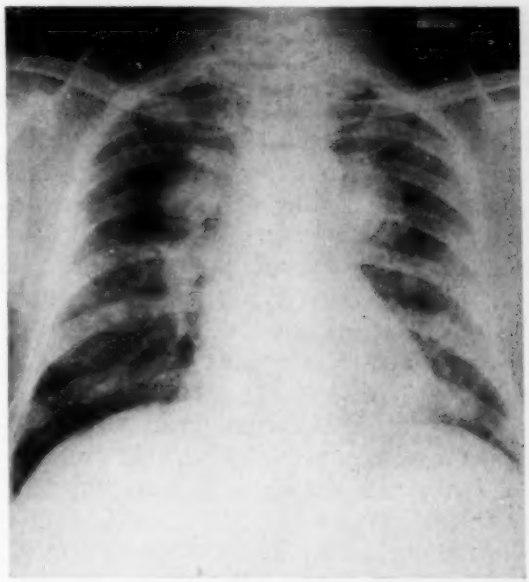


Fig. 14. *Possible manifestation of silicosis.* J. E. M., male, aged sixty-six. This individual has been working in sand pulverizing for a number of years and about the only evidence of anything abnormal is the tremendous shadow of the tracheobronchial lymph nodes in the hila. I have studied this patient for at least seven years and the hila have not changed. The patient has practically nothing abnormal in the lung fields and is not disabled in any way. We of course thought that he might have a childhood type of tuberculosis, a lymphoblastoma of some type or some other unidentified condition such as Boeck's sarcoid. There is no nodulation in the lung field. I have found 9 such cases in this particular industry and one wonders whether or not this may be a peculiar manifestation of silicosis in which the marked silicotic changes are being manifested in the tracheobronchial lymph nodes rather than elsewhere.

Another explanation is that the scar contraction of the silicotic process may be sufficient to produce the conglomerate lesion.

The shadow of the conglomerate lesion in simple silicosis is often difficult to distinguish from that found in silicosis with infection. From the roentgenologic standpoint they may be impossible to differentiate in a single examination, whereas serial examinations may or may not show slight changes in the extent of the shadows of lesions that are complicated by an active infection.

The roentgen observations in *simple silicosis* may be summarized as follows:

Trachea: Its shadow is in a normal position.

Heart and aorta: Their shadows are not noticeably affected by the silicotic process.

Domes of the diaphragm: The shadow and movement of the domes of the diaphragm are not affected by the silicotic process unless a coincident emphysema is present.

Hila: The shadows of these structures are as a rule not noticeably changed. In some instances they seem large (Fig. 14).

Trunk shadows and linear markings: Slight to moderate variations in these shadows are difficult to evaluate.

Lung changes, nodulation and conglomerate lesions: The shadows of *nodulation* (2–6 mm. in size) are usually bilateral in distribution. Variations may occur such as a lobar or unilateral distribution.

Shadows of nodules may be difficult to differentiate from those occurring in some types of tuberculosis, metastatic malignancy and fungus infestation, etc.[28]

Conglomerate lesions: These shadows are usually found in the upper half of the lung fields and are difficult to evaluate in single examinations. Superimposed metastatic malignancy and lesions due to an active infection may produce similar appearances.

Hyperventilation and emphysema: Such conditions may be present and demonstrable by the roentgen examination. Their evaluation, as to cause and effect from dust exposure, however, is more difficult and does not fall within the province of the roentgenologist.

SILICOSIS WITH INFECTION

In this group are included all cases of silicosis with detectable evidence of infection. It is not always possible in the living patient to determine whether the infection is active or inactive even in instances when conventional clinical and laboratory examinations are available. Under such circumstances, mistakes will occur, but if one exercises good judgment, the affected individual can be protected by taking the necessary precautions.

The lesions (either some or all) that have been described as occurring in simple silicosis may be modified by infection. Other lesions that may be found include cavities (usually thick walled) tuberculous in origin, cavities occurring as a result of necrosis of anemic infarcts, massive lesions, mottling, soft nodulation, various degrees of emphysema and bleb formation, pleural thickening, pleural collections, pneumothoraces and deformations of the domes of the diaphragm. The roentgenologist is rarely able to predict whether the infection is due to the tubercle bacillus or some other organism. One suspects, however, that in the majority of instances, the superimposed infection is tuberculosis, for Gardner[10] states that the postmortem examinations showed an element of tuberculosis in 60 per cent of the cases.

In order to obtain the maximal amount of information from the roentgen examination, one must occasionally include over and under exposed stereoscopic roentgenograms made in the anteroposterior and posteroanterior positions, oblique and lateral views, films exposed with the use of the Potter-Bucky diaphragm, body section roentgenograms exposed in the erect and horizontal postures, and films exposed in each lateral decubitus.

The changes occurring in the various structures of the respiratory and cardiovascular tracts as demonstrated by the roentgen examination may be summarized as follows:

Trachea: Its shadow may be in a normal position, especially if a tuberculous process or some other infection is superimposed

upon an already established silicotic process. In some instances the trachea is found displaced to one side (Fig. 18*A* and *B*). At such times, it is my feeling that the silicotic process has occurred either simultaneously with tuberculosis, or the tuberculous lesion was probably established before the silicotic changes became manifest, or the traction of the fibrotic changes produced by a tuberculous and silicotic lesion on one side was greater than that of the silicotic process of the opposite side.

Heart and aorta: In some instances evidence of cor pulmonale is observed in the advanced silicotic individual. Some clinicians[6] have regarded this condition as a complication of silicosis. The relation between cause and effect is difficult to establish in this instance and cardiologists who have given some thought to the question find themselves unable to express an opinion. Many of us who have been interested in silicosis for years have not been impressed with the incidence of cardiac complications. On the other hand, it is possible that with increased use of the electrocardiograph in the well advanced silicotic, more evidence of slight heart changes may be found. But even if such evidence is found, it is well known to the cardiologist and the general pathologist that in the older age group not exposed to harmful dust, which come to autopsy, cardiac complications are not uncommon. Even in the advanced silicotic with well established emphysema, Gardner[10] states that "hypertrophy of the right side of the heart without arteriosclerosis and even greater involvement of the opposite side is a rarity."

Occasionally the heart and aorta are displaced to one side or backward by the contracting scar of a tuberculous infection and silicotic process. As in the case of the trachea and in the absence of data to the contrary, such a displacement is thought to occur for the same reasons.

Domes of the diaphragm: If considerable emphysema is present, the domes of the diaphragm may be depressed and limited in their movements. At times it is necessary to have the patient cough in order to demonstrate, roentgenoscopically, evidence of diaphragmatic excursion.

Individualization of the costal components of the domes of the diaphragm is quite marked when there is considerable basal emphysema.

Another abnormal appearance is multiple peaking of the domes of the diaphragm. It may be impossible to differentiate the peaking caused by pleural adhesions from that due to inelasticity of certain structures of the lung. Both conditions may be present.

Hila: The shadows of the hila may or may not be within normal limits. The hilum shadow may be enlarged or its shadow may be partly or totally obscured by a larger shadow produced by a lung lesion. The hilum may be displaced upward, laterally or backward by a contracting scar of a silicotic process plus an infection (similar to that causing displacement of the trachea).

Bronchial tree, lymphatic and vascular channels with their supporting structures: Oftentimes the otherwise increased prominence of these structures is obscured by shadows of parenchymal lesions, therefore the evaluation of trunk shadows and linear markings rarely play a part in helping one arrive at a correct diagnosis. The presence of bronchiectasis is difficult or impossible to detect in many instances.

Lung changes: Mottling is a term that is applied to the small and poorly defined shadows produced by an acute or chronic infection. The roentgen manifestations of mottling and nodulation may be identical and exceedingly difficult or impossible to differentiate. The shadows of mottling often lack a uniformity of distribution and are more likely to change in form in subsequent examinations than are the shadows of nodules. Mottling usually occurs as a result of a bronchogenic spread of a tuberculous lesion or the result of a fungus infestation.

Soft nodulation: The shadows of soft nodulation are much larger than those produced by nodulation and mottling, but are smaller than the conglomerate shadow (Fig. 15*A* and *B*). The description of such a roentgenologic shadow is provided in order to emphasize a perinodular cellular reaction

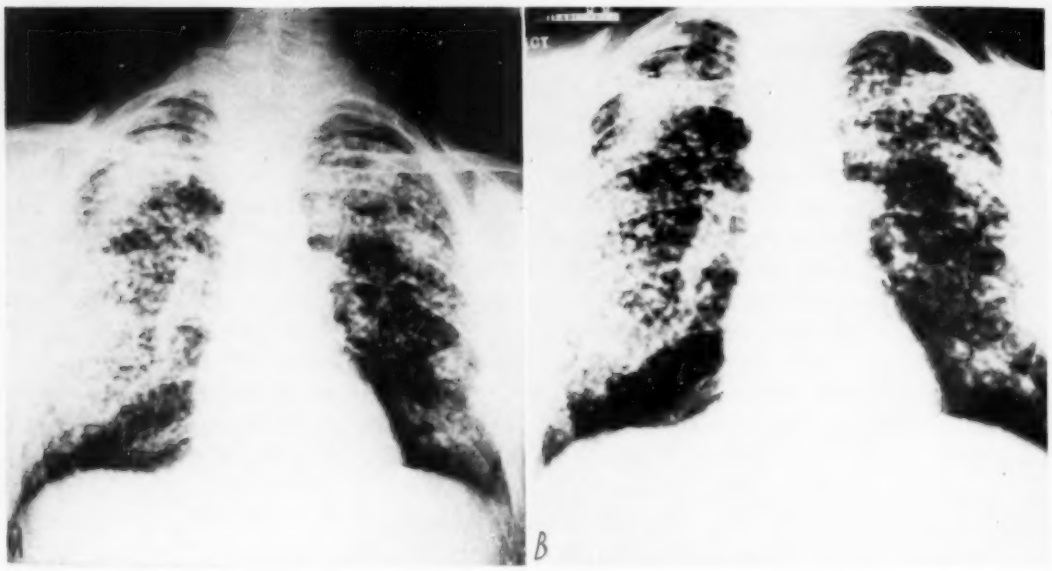
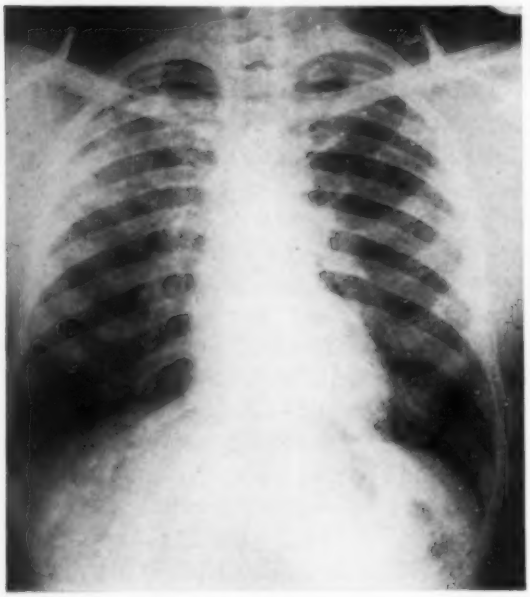


Fig. 15. *A. Anthracosilicosis with infection.* J. T., male, aged forty-three, who came into the hospital with a diagnosis of brain tumor. He had a history of working in a hard coal mine for thirteen years and in a slate mine for four years. The case is shown to illustrate large nodule formations which may represent the soft nodulation that occurs in silicosis with infection. There are conglomerate shadows in the upper lobes. *B*, roentgenogram made with the use of the Potter-Bucky diaphragm showing the soft nodulation to better advantage.

that is observed by the pathologist. These shadows are much more likely to change in character in subsequent examinations than is the shadow produced by nodules, and change less than those due to mottling.



Nodulation: The shadows of nodulation are similar to those described under "simple silicosis." Soft nodulation and nodulation cannot be demonstrated by a roentgen examination in every case of modified silicosis with infection. This is particularly true in certain industries such as hard coal mining and granite cutting. In cases where there is only roentgen evidence of massive or conglomerate shadows one is more likely to make errors in interpretation.

Fig. 16. *Silicosis with infection.* V. H. J., male, colored, aged forty-six. First examination October 21, 1938. Various jobs from 1916 to 1933 which are probably of no significance. In 1933 he began to do sand blasting and in a survey examination on October 21, 1938, the roentgen findings, illustrated above, show diffuse nodulation in the left lung field and a similar appearance on the right side, except that in the right upper half of the lung field the nodules are somewhat conglomerate. The patient had no symptoms of disability and did not have any elevation of temperature. I felt at that time, however, that he had a well advanced silicotic process with probable superimposed infection. See Figure 17.

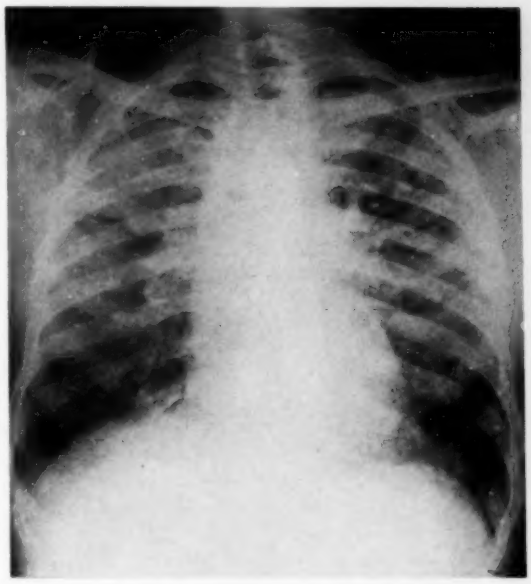


Fig. 17. Same case as Figure 16. Examination made February 20, 1942. At this time the silicotic process has advanced and there is now a mass of consolidation in the right upper lobe, slight deviation of the trachea toward the right side with conglomerate nodulation in the right lower lobe and in the left lung. There is a moderate amount of hyperventilation in the lower lung fields. Since the first examination the individual continued to work but, at this time, he is quite short of breath and he thought that he had influenza. His condition is really one of silicosis with infection and clinically he was thought to be totally disabled. See Figure 18.

Massive shadows: These shadows vary from 3 to 20 cm. in size. Some are round, some oval and some wedge shaped. These lesions are usually due to extensive areas of fibrosis and are found in the upper third or upper two-thirds of the lung field (Fig. 16–18*B*). Occasionally it is possible with overexposed films, body section roentgenograms or a Potter-Bucky roentgenogram to show some of the details of a massive lesion, such as distorted and obliterated bronchi, cavities and areas of calcification and caseation.

A few years ago, most of these lesions were thought to be due to silicosis with infection. Riddell,[31] Gardner,[10] McCloskey[22] and others question whether this is always true. Gardner[10] thinks that a "third essential factor may be minerals other than free silica."

From a roentgenologic standpoint, I have attempted to determine whether the massive lesion extended to the periphery of the involved lobe of the lung and have used

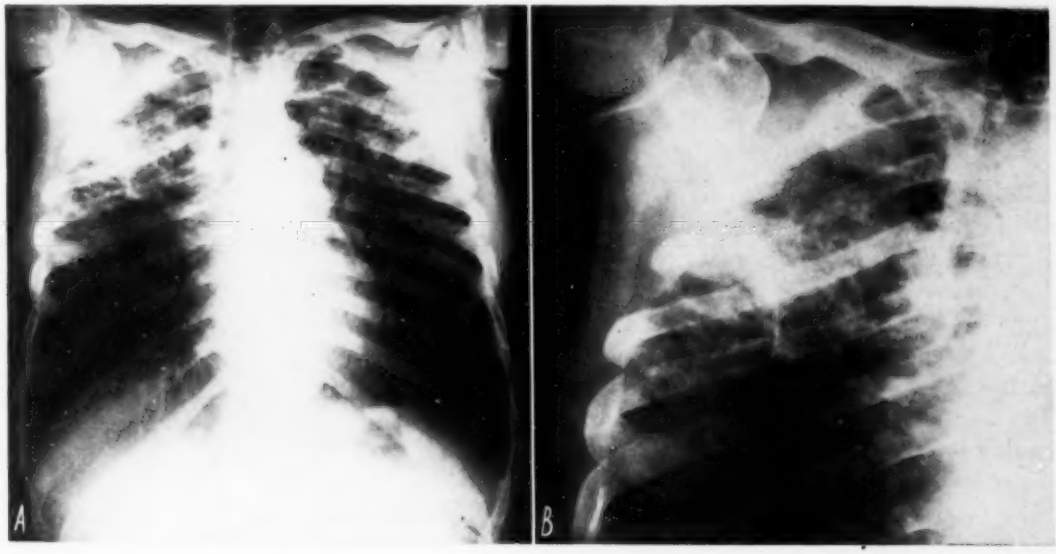


Fig. 18. *A. Silicosis with infection.* Same case as Figure 17. This illustration is a roentgenogram made with a Potter-Bucky diaphragm showing the massive shadows in the lung and the position of the trachea to better advantage. *B.* Close-up of the right upper lobe showing the detail of the intrapulmonary process.

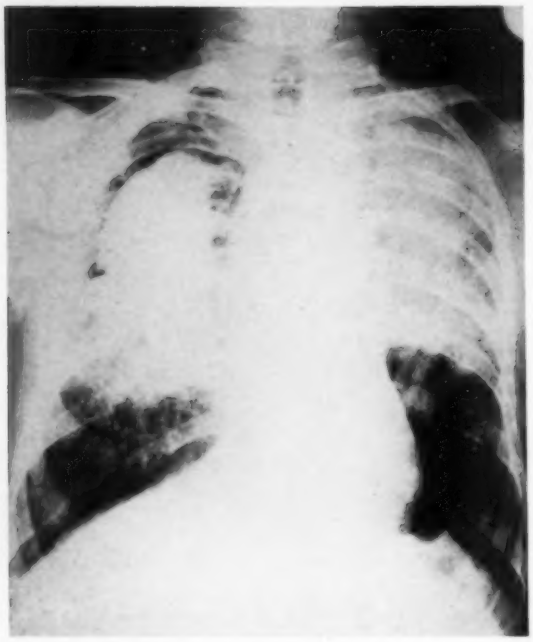


Fig. 19. *Anthracosilicosis with massive lesions and active infection.* M. B., male, aged fifty. This patient had worked as a hard coal miner since he was twenty-one years of age. He has done dry drilling all the time and has never used a mask. He stopped work one year ago because he was unable to carry on. The roentgen examination, at that time, showed a massive lesion in each upper lobe. There was a pneumothorax on the right side. Note that the lesion extends out to the periphery but it was not adherent. Another interesting observation is the absence of any roentgen evidence of nodulation outside of the massive lesion. The lesions are similar in extent in both upper lobes. It was felt that these lesions contained an active infection because they extended to the pleura. There is evidence of emphysema in the lower lobes.

this information as evidence for or against the presence of an active infection. For instance, if a lesion extends to the interlobar fissure or the thoracic wall, such a lesion would be regarded as the seat of an active infection (Fig. 19 and 20). If the massive lesion were situated in the center of the affected lobe, the roentgen evidence would not necessarily be in favor of an active infection (Fig. 21). I realize that such deductions by themselves are of very little value, but in the absence of more convincing clinical and pathological proof attention to this detail may prove to be helpful.

The massive lesions do not tend to displace the midline structures (trachea, hila, heart and aorta) by scar contraction. When displacements of those structures occur, the lesion which produces them is more likely to be either a tuberculous or Friedländer bacillus infection which has preceded or occurred simultaneously with the silicotic process. It should be possible, however, for the contracting scar of a tuberculosilicotic process of one lung to overcome that of an uncomplicated silicotic process on the opposite side.

Occasionally, one sees a lesion which by roentgen methods is diagnosed as a massive lesion, which at autopsy turns out to be a necrotic, anemic infarct or a cavity in which there is no microscopic evidence of infection and which is filled with a syrup-like material (Fig. 22). I know of no way to diagnose the true nature of these lesions by the roentgen examination.

Tuberculous cavities may occur in the upper or lower lobes. If the cavity is open,

Fig. 20. *Anthracosilicosis.* The bilateral massive lesions are probably due to anthracosilicosis with a superimposed infection. There is extensive emphysema in the bases. This man, a coal miner, was working up to and after the time that this examination was made.

there may be a bronchogenic spread to other portions of the lung. The cavities as a rule are thick walled. Oftentimes, even in the presence of acceptable roentgen evidence of active disease, the clinical manifestations are either absent or confusing. Dyspnea, vague chest pains and some loss of weight may be the only symptoms present. Oftentimes it is impossible to obtain positive sputum.

The terms tuberculosilicosis and silicotuberculosis as used by us have different meanings. The term tuberculosilicosis is reserved for those cases of tuberculosis complicating a silicotic process in which there is some displacement of the midline structures (trachea, etc.) whereas the midline structures are not displaced in silicotuberculosis.

In South Africa, tuberculosilicosis is a lesion in which the dust factor is dominant,

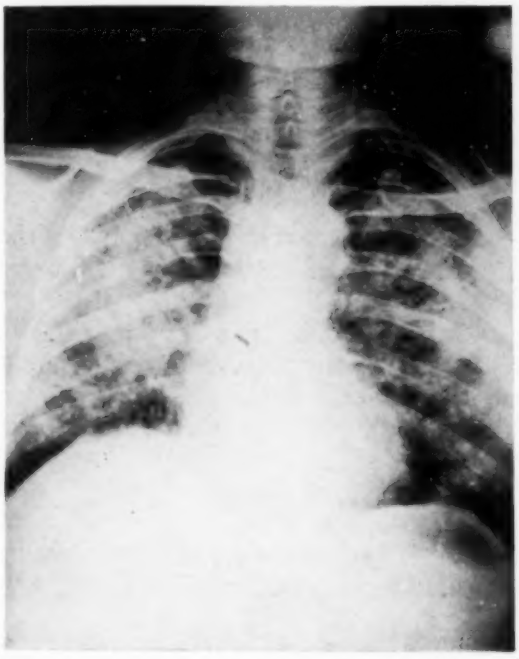


Fig. 21. *Anthracosilicosis.* T. P., male. This man was a hard coal miner for a number of years. He has a lesion involving both lungs. The lesion is manifested by conglomerate and beginning massive shadows and some nodulation. The heart shadow is normal. The trachea is in the midline and there is relatively little roentgen evidence of emphysema in spite of the tremendous lesions.

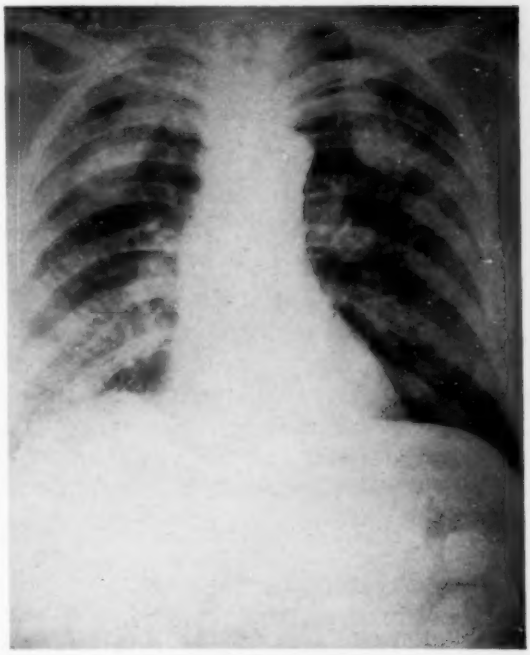


Fig. 22. *Anthracosilicosis with massive shadows in each upper lobe.* S. R., male. This man was a coal miner for several years and films of his chest show massive lesions in the upper lobes and elsewhere there was evidence of nodular lesions. The nodules are ill defined. At the postmortem examination the two shadows in the upper lobes were due to anemic infarcts with aseptic necrosis. No evidence of the cavities can be seen by this examination. Careful microscopic studies of the tissues failed to reveal any evidence of infection. It was therefore felt that the lesions were anemic infarcts with cavity formations rather than those due to infection. (Illustration used by courtesy of Dr. Bernard McCloskey.)

and in silicotuberculosis the infectious element is more prominent.

The massive lesion occurring in silicotic individuals is fairly typical especially if shadows of nodules can be seen elsewhere in the lung. When there are no shadows of nodules visible, the diagnosis becomes more difficult, and its differentiation from other lesions producing similar shadows depends on the history and the physical and roentgen examinations.

Emphysema and bleb formation: Emphysema frequently, and in some instances bleb formation, is observed in silicosis with infection. By roentgen methods, the emphy-

sema is noted particularly in the bases, along the periphery of the lobes, and in the apices. These are the areas where the nodules are less likely to be seen. Bleb formation is noted particularly peripheral to the shadows of massive lesions.

Pleural abnormalities: If a spontaneous pneumothorax develops in a patient one can readily see the shadows of thickened visceral pleura (Fig. 19). In some of the silicotic patients with pneumothorax an increased respiratory embarrassment out of proportion to the amount of air in the pleural cavity is manifest. Such experience makes one question the advisability of using pneumothorax as a therapeutic procedure in silicosis with active pulmonary tuberculosis.

Pleural thickening may occur in the interlobar fissure, along the visceral pleura and may cause considerable peaking of the domes of the diaphragm. Pleural adhesions may obliterate the usual costophrenic sulci. Pleural collections manifest themselves roentgenologically just as they do in the nonsilicotic.

PNEUMOCONIOSIS AND SILICOSIS OCCURRING IN VARIOUS INDUSTRIES

All of the roentgen manifestations that have been described above have taken into consideration the hypothesis that the lesions in many or all instances have been produced by free silica and modified by other dusts (contaminants). As stated before, it is probable that very few opportunities are afforded to study lung changes in which pure silica is the only dust present. One should therefore bear in mind that the manifestations of silicosis in the various industries are likely to be different. For instance, the roentgen findings in the slate and talc workers are likely to be different from those occurring in hard and soft coal miners. The roentgen findings may differ even in different foundries. In most instances it requires many years (six to fifteen) of exposure before one can demonstrate roentgen evidence of silicosis. The exceptions to this rule are classified under the heading of "rapidly developing silicosis."

Rapidly developing silicosis has been described by Gardner.[12] When individuals are exposed to excessive quantities of finely divided silica, an unusual kind of reaction develops. The overwhelming deposits of silica seem to stimulate connective tissues in every part of the lung at the same time. The nodules are of microscopic proportions and in most cases there have been complicating infections.

I have seen a great many roentgenograms (some single, a few stereoscopic) of individuals who were diagnosed as having rapidly developing silicosis. In the advanced cases, lobar consolidations due largely to the superimposed infection obscured the changes due to silicosis. In the earlier cases the roentgenographic manifestations were those sometimes observed in an interstitial type of pneumonitis of ill defined shadows similar to that produced by a prominent pectoral fold. In a few cases in which I made roentgenoscopic observations, the costal and diaphragmatic excursions seemed to be limited. A number of the individuals that I saw had lost weight, were pale and dyspneic. Experience in this country was similar to that in England,[23] in that most of the fatal cases died with tuberculosis.

Another consideration having to do with silicosis is concerned with what might be called individual susceptibility. Physicians interested in this subject have noticed that not all individuals exposed to dust in the same industry, and apparently in the same manner, develop silicosis. Why such a difference is manifest no one knows. Many explanations have been offered, but none of them are completely satisfactory. Lanza[18] feels that the question of individual susceptibility has been overworked and is not as significant as the question of dust exposure (dosage). There seems to be a general agreement that when a tuberculous infection is present, the action of the silica and the infection are enhanced.

There are many avenues of research con-

cerning pneumoconiosis still to be explored. Every year brings new answers to old problems and new problems to be answered. Until we know the answers to all of the problems concerned with this subject it behooves us to plan for the worker. Such a plan should include the following requirements:

1. A healthy atmosphere in which individuals have to work.

2. Pre-employment and annual physical and roentgen examinations of the chest in order to demonstrate early tuberculous lesions and exclude such individuals from participating in work that may be harmful to them and their fellow workers.

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ANATOMIC-ROENTGENOLOGICAL ANALYSIS OF THE NORMAL HILAR SHADOW*

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CONCEPTION OF THE TERM. PRELIMINARY CONDITIONS OF THE ANALYSIS

THE idea of a hilar shadow was conceived of in the beginning of the era of roentgen diagnosis. Obscure anatomical ideas apparently determined the choice of the name. For the *hilus* according to anatomical definitions is a superficial zone (a "portal of entry," a "plane of cleavage"), with almost linear cross-section. It cannot in itself possibly be the only substrate of the broad shadow that is known by the name of "the hilar shadow."

Later, various authors showed that all the constituents of the root of the lung, but particularly the vessels, form the foundation of the above-mentioned shadow. It would therefore be more correct to call it the "root shadow." But it is hardly possible now to change the old established name. But it must be understood that the roentgen anatomy of the hilar shadow is the roentgen anatomy of the root of the lung.

The analysis of the hilar shadow is based on accurate examinations of individual cases. General knowledge, that is knowledge that can be applied to other cases, is deduced from these results. This object can be attained only when (*a*) the anatomical substrate presents sufficient uniformity and regularity and (*b*) a rule for the objective boundaries and description of the root of the lung can be found.

(*a*) The form of the normal hilar shadow is determined chiefly by the vascular system. Therefore the existence of a sufficient regularity in the vessel architecture is the first requisite for the success of the work. The textbooks of anatomy do not give sufficient information on this point. Only a few special works on the subject are to be found and their reports are contradictory. Therefore it was a necessary preliminary task to study the normal anatomy of the pulmonary vessels with special attention to the details that concern roentgenology. I did this in collaboration with Kubat.[5]

We found in agreement with Ewart,[1] Melnikoff,[6] and others, that the anlage and development of the vessels in the lungs, as elsewhere in the body, show constancy in the essential points. This is manifested in a very great similarity in the number, arrangement, direction, point of origin and region supplied by all the chief vessels and their typical branches. The same thing is true of the bronchi, which in the lungs are completely correlated with the arteries. There is also an extensive parallelism between the right and left lungs, the lingula being the homologue of the middle lobe. Therefore it will be sufficient if in the following pages I discuss only one lung, the right one.

There are of course many and manifold variations in the different vessels and differences in the type of branching. These individual variations however are of subordinate importance. (For details and bibliography see Herrnheiser and Kubat.[5])

The parallelism between the middle lobe and the lingula was first recognized by Ewart. It supplies the key to the correct understanding of the architectural plan of the vessels of the lung. Certain differences between the right and left lungs are of no practical consequence.

A natural arrangement of the root of the lung can be deduced from the description of the lobes of the lung given by the anatomist Felix. According to Felix[2] every lung lobe consists of a "lobe mantle" and a "lobe nucleus." The "lobe pedicle" originating from the lung pedicle passes into the latter.

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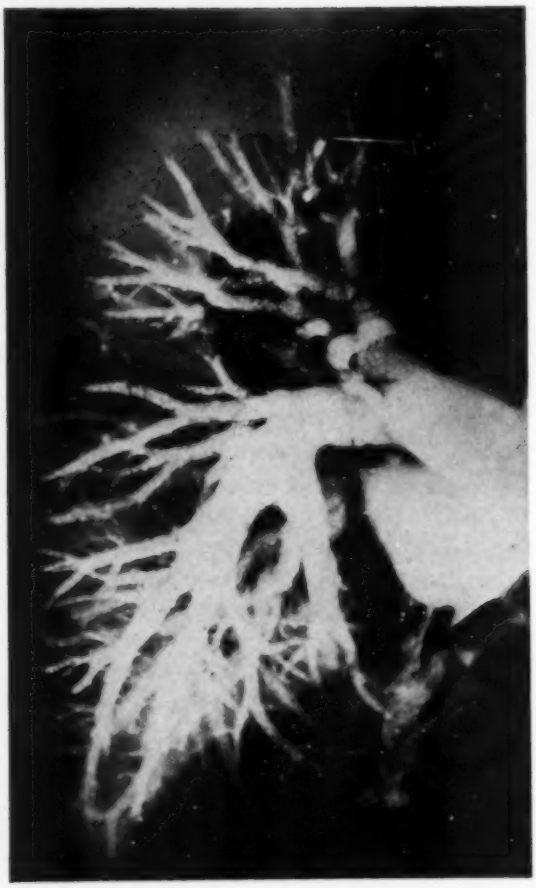

Fig. 1. Injection roentgenogram of the right lung. The nucleus of the upper lobe has been dissected.

The branching of the lobe pedicle takes place in the "inner zone of the lobe nucleus." But in the latter the spaces between the vessels and the bronchi are still compactly filled with connective tissue and lymph nodes. It is only further toward the periphery in the "outer zone" of the lobe nucleus that larger lung lobules push between the diverging branches of the vessels and of the bronchi.

We shall consider the inner zone of the lobe nucleus as the intrapulmonary segment of the root of the lung. Together with the lobe pedicle belonging to it, it constitutes a "lobe root" The root of the lung can therefore be divided anatomically into the lobe roots and the lung pedicle formed by their union.

The difficult task of establishing an anatomical boundary of the root of the lung is also settled by the above-described division of the lobes by Felix. The peripheral boundary is naturally to be placed where the closed bunch of vessels and bronchi impinges on the air-containing parenchyma. This takes place at the boundary between the inner and outer nuclear zones. This boundary generally runs over the secondary branches of the bronchi, often near or at their tertiary division, and is marked by the branching of very fine bronchi supplying the first lobules, the so-called "rudimentary nuclear lobules" (Felix). The vessels also often show an analogous boundary line, namely the appearance of the first "small nuclear lobular branches." I have described the latter in a paper before the Fourth International Congress of Radiology in Zurich.[4] (For greater detail see Herrnheiser and Kubat.[5])

Technique of Examination

The analysis of the hilar shadow was made from roentgenograms of injected lungs of cadavers. Figure 1 shows such a roentgenogram. We had available injection roentgenograms of 41 lung preparations of our own; the majority of these, however, were suitable only for purposes of comparison. The technique of preparing the specimens and the varying nature of their elaboration is discussed by Herrnheiser and Kubat. The detailed analysis described here which relates all shadows to their anatomical substrate, I was able to carry out on 6 lungs.

In identifying the vessels I made use of photographs which were taken during and immediately after the injection of the expanded lungs. First, the vessels of the upper lobe were dissected in layers, beginning in front, every phase of the dissection photographed and, after as complete reconstruction as possible of the normal site, roentgenographed (see Fig. 1). The exposed vessels were identified anatomically and the corresponding stripes of shadow located in the roentgenograms. Questionable ves-

sels are marked with different kinds of bits of lead and so definitely defined in new roentgenograms. The results are marked in sketches accurately traced from the roentgenograms. Then the vessels in the middle and lower lobes were exposed in the same way, photographed and identified.

The examination was continued until there was absolute certainty in regard to the substrate of each of the larger vessel shadows. Finally in this way we obtained an identifying sketch of all the vessel shadows involved, which agreed absolutely with the roentgenogram. Figure 9 shows such a sketch. This comes from the same specimen as Figure 1, as well as all the other illustrations in this publication.

I had to refrain from giving a simultaneous representation of the vessels and bronchi, because the roentgenograms made after simultaneous injection of the two systems were not clear.

ANATOMY OF THE VESSELS

The "trunk" of the pulmonary artery is, according to Assmann and others, the chief constituent of the hilar shadow. We must examine the correctness of this statement. Our designations of the sections of the pulmonary artery vary, however, in some respects from the official anatomical nomenclature. I would like, therefore, to discuss them briefly with reference to the illustrations and the arguments in Herrnheiser and Kubat.

The true trunk of the pulmonary artery ends in the mediastinum with a division into two large branches: the superior and intermediary trunks.

The superior trunk (*Tr. s.*) constitutes the main artery of the upper lobe. As a rule, it enters the nucleus of the upper lobe undivided. Not infrequently, however, it branches even outside the lung into the three upper lobe arteries of the first order (the apical artery, the pectoral artery and the axillary artery).

The intermediary trunk (*Tr. im.*) carries blood to the middle lobe and the lower lobe. It begins at the bifurcation of the pulmonary artery, bends downward and ends in front with the giving off of the artery of the middle lobe (A. lobi medii) and behind with the branching off of the uppermost artery of the first order of the lower lobe (A. apico-horizontalis), which generally lies higher up.

The inferior trunk (*Tr. i.*) is the continuation of the intermediate trunk and forms the chief arterial constituent of the lower lobe root. It begins where the intermediate trunk ends. After a course of varying length, it divides finally into a ventral and a dorsal pair of arteries of the first order directed toward the base (A. basilaris and A. axillo-basilaris anterior and A. basilaris and A. axillo-basilaris posterior).

The Arteria lobi medii is the chief artery of the middle lobe. It also from its character holds the rank of a trunk. But in the abovementioned work with Kubat I have kept the old nomenclature and will not change it, in order to avoid misunderstanding. It is sufficient to know that the two branches of the A. lobi medii—R. paramediastinalis and R. costalis—are to be considered as lobe arteries of the first order, the more so as they not infrequently spring separately from the intermediate trunk.

The pulmonary veins also are involved in the morphogenesis of the hilar shadow

The upper pulmonary vein as a rule consists of three branches (rami). Among these the posterior ramus (R. posterior) is the largest and to a certain extent the chief trunk. It is divided into two lobe veins of the first order (vena apicalis and vena axillaris). The two other branches—R. superior and R. inferior—are roentgenologically of less importance.

The lower pulmonary vein has two branches, the superior and inferior branches. The ramus superior continues in the lower lobe as a vein of the first order (V. apico-horizontalis). The ramus inferior is a large branch from which the basilar lower lobe veins originate.

But numerous other vessels of the first and second order lie in the region of the root of the lung which must be taken into

TABLE I

SYSTEM OF PULMONARY VESSELS TOGETHER WITH A GENERAL EXPLANATION OF THE DESIGNATIONS OF THE VESSELS IN ALL FIGURES

The Chief Trunks of the Pulmonary Vessels and the Lobe Vessels of the First and Second Order

Arteries	*Veins*
	Chief Trunks
A.p.d. A. pulmonalis dextra	Upper pulmonary veins
Tr.s. Truncus superior	Ramus superior
Tr.im. Truncus intermedius	*R.p.* Ramus posterior
	Ramus inferior
Tr.i. Truncus inferior	Lower pulmonary veins
	Ramus superior
	R.i. Ramus inferior
	Upper Lobe Vessels
	V 1 V. subpleuro-mediastinalis
	V 1a R. apico-mediastinalis
	V 1b R. sterno-mediastinalis
A 1 A. apicalis	*V 2* V. apicalis
A 1a R. mediastinalis	.....
A 1b R. costovertebralis	*V 2b* R. apico-centralis
A 1c R. costalis	*V 2c* R. costalis
A 2 A. pectoralis	*V 3* V. pectoralis
A *2a* R. sterno-pectoralis horizontal.	.....
A 2b R. sterno-pectoralis descendens	
A 2c R. costo-pectoralis ascendens	.....
A 2d R. costo-pectoralis descendens	.....
A 3 A. axillaris	*V 4* V. axillaris
	Middle Lobe Vessels
A 4 A. lobi medii	
A 4a R. paramediastinalis	*V 5* V. paramediastinalis
A 4b R. costalis lobi medii	*V 6* V. costalis lobi medii
A 4bα R. costalis axillaris	*V 6a* R. costalis axillaris
A 4bβ R. costalis descendens	*V 6b* R. costalis descendens
	Lower Lobe Vessels
A 5 A. apico-horizontalis	*V 7* V. apico-horizontalis
A 5a R. retrobronchialis	*V 7a* R. retrobronchialis
A 5b R. apicalis	*V 7b* R. apicalis
A 5c R. horizontalis	*V 7c* R. horizontalis
A 6 A. basilaris anterior	V. basilaris anterior [often lacking]
A 6a R. descendens	
A 6b R. angularis	
A 7 A. axillo-basilaris anterior	*V 9* V. axillo-basilaris anterior
A 7a Rr. axillares ant.	*V 9a* Rr. axillares ant.
A 7b Rr. basilares ant.	*V 9b* Rr. basilares ant.
A 8 A. basilaris posterior	*V 10* V. basilaris posterior
A 9 A. axillo-basilaris posterior	V 11 V. axillo-basilaris posterior
A 9a Rr. axillares post.	
A 9b Rr. basilares post.	

consideration in the analysis of the hilar shadow. It would certainly be advantageous in the understanding of further descriptions if a more accurate anatomical description of these vessels could be given. But that is not possible within the limits of

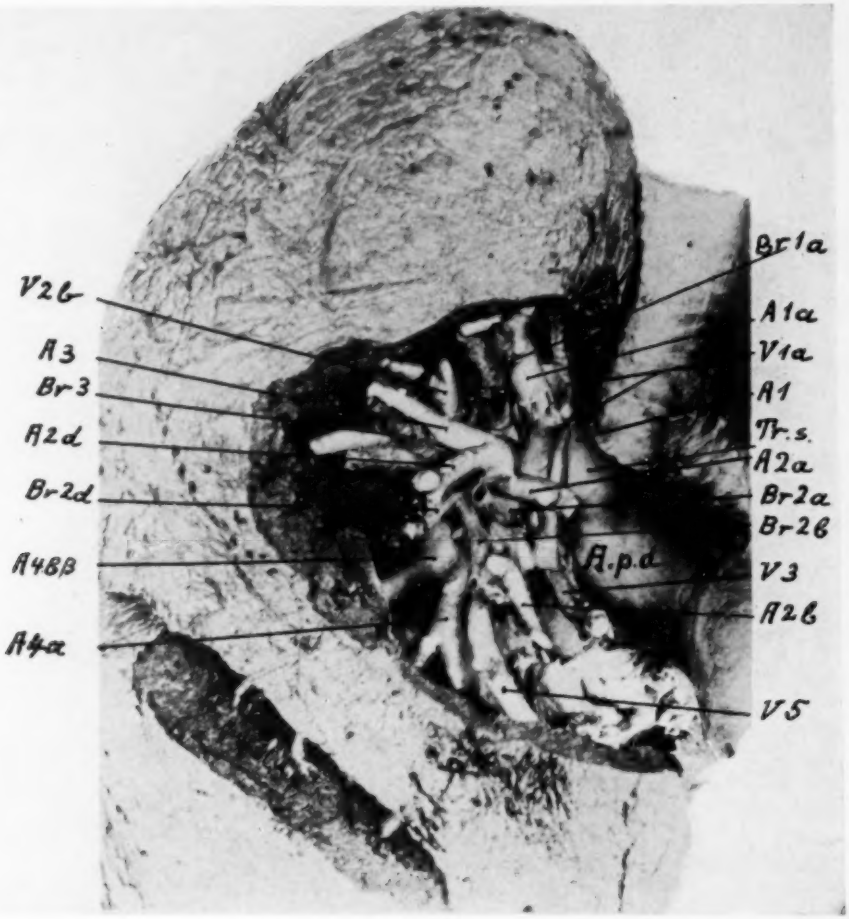

Fig. 2. Right upper lobe root, anterior view. In the lower part of the picture some middle lobe vessels. *Description:* In dissecting, the secondary branches of the arteries and bronchi of the ventral region of the lobe are met with first. These are branches of the pectoral artery and bronchus: medially the R. sterno-pectoralis horizontalis (*A 2a*, *Br. 2a*) and the R. sterno-pectoralis descendens (*A 2b*, *Br. 2b*); laterally the R. costo-pectoralis descendens (*A 2d*, *Br. 2d*). In the anterior lobe region lie also the V. pectoralis (*V 3*) and the R. apico-mediastinalis (*V 1a*) of the V. subpleuromediastinalis. The central root region is for the most part covered by these vessels. But structures of the deeper layers are also visible, such as: medially the superior trunk (*Tr. s.*), the apical artery (*A 1*) originating from it, and its R. mediastinalis (*A 1a*). Further laterally in the cranial part of the exposed region the R. mediastinalis of the apical bronchus (*Br. 1a*), and also the R. apico-centralis of the apical vein (*V 2b*) come into view. Also the axillary artery and bronchus (*A 3*, *Br. 3*) are distinctly recognizable. The middle lobe vessels can be seen better in Figure 3.

this work. Therefore only a tabulated summary (Table 1) of the primary and secondary lobe vessels must serve as a foundation for the later special description.

This table serves also as a general explanation of the designations. Each vessel in the illustrations in this paper is always marked with the same letters and the same figure. These designations are also used in the table, so that for every designation the name of the vessel can easily be found. The designations are also identical with those in the work of Herrnheiser and Kubat.

Roentgen Anatomy of the Root of the Lung

The roentgen picture of the root of the lung, as a whole, shows such a fullness of shadow detail that orientation is difficult even after the vessels have been identified.

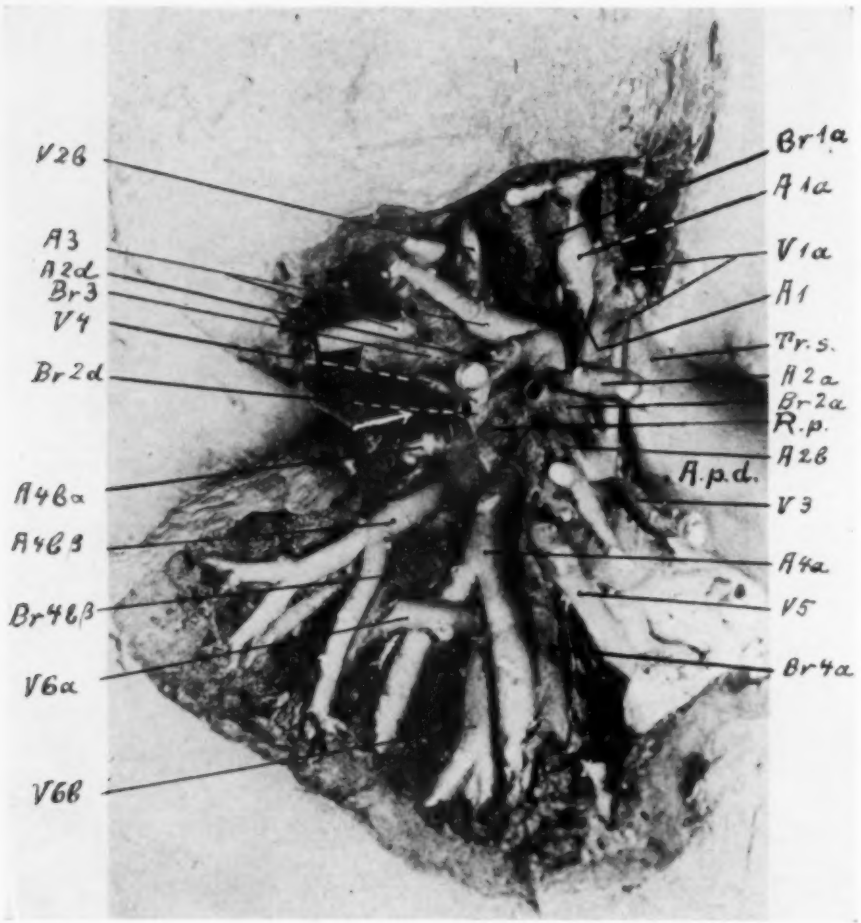

Fig. 3. The central part of the upper lobe root. The middle lobe root entirely exposed. *Description:* The ventral branches of the vessels and bronchi (*A 2ad*, *Br. 2ad*, *V 3*) are drawn medially and upward with threads. In this way the R. posterior of the upper pulmonary vein (*R. p.*) and the axillary vein (*V 4*) originating from it become visible. Also the R. apico-centralis (*V 2b*) of the apical vein now becomes more distinctly visible. The other upper lobe vessels as in Figure 2. The angle of intersection (→) of the R. posterior with the Tr. intermedius of the pulmonary artery, open laterally, can also be seen. Below the arrow the mediastinal end of the fissure between the upper and middle lobes can be seen in the vertex of the angle of intersection. The middle lobe root is dissected extensively. The R. costalis-axillaris (*A 4b α*) and the R. costalis descendens (*A 4b β*) spring here as variants separately and directly from the Tr. intermedius, instead of uniting with the R. paramediastinalis (*A 4a*) to form the common trunk of the A. lobi medii. The V. paramediastinalis (*V 5*) is visible almost in its entirety. The V. costalis lobi medii is partially covered but its branches, the R. axillaris (*V 6a*) and the R. ascendens (*V 6b*), are visible. Special attention should be paid to the R. axillaris as a boundary mark.

I will therefore in my presentation almost repeat the course of the anatomical analysis: that is, I will first discuss the roentgen anatomy of the individual lobes of the lung and on the results reconstruct the analysis of the whole picture (Fig. 9).

The anatomical conditions are illustrated by the figures of the phases of the dissections (Fig. 2, 3, 6 and 7). The detailed description of the figures should suffice for explanation. The roentgenograms of the lobe roots are represented by accurate tracings of the corresponding parts of the injection picture in Figure 1. The complete chart of the identified vessels (Fig. 9) is made up of these partial sketches (Fig. 4, 5 and 8).

In the roentgenograms the boundary and

the structure of the root region are particularly interesting. The anatomical boundary surfaces appear in the picture in part as lines. But where the projection of the boundary surfaces is broader and more complicated I have simplified by assuming a "middle boundary line" as a resultant. The boundary from the air-containing lung tissue I have designated as a "peripheral boundary." As the anatomical boundary from the parenchyma is uneven this is always represented as a middle boundary line.

a. The Right Upper Lobe Root (*Fig. 2, 3 and 4*). The chief structures of the upper lobe root are: the Tr. superior of the pulmonary artery (*Tr. s.*), the eparterial bronchus and the rami of the upper pulmonary vein, especially the posterior ramus (*R. p.*). These, together with their primary and a large part of their secondary branches, form a fairly large closed complex (Fig. 2). The vessels and bronchi lie at different depths in this, so that the anterior vessels must be drawn aside in order to see the central parts (Fig. 3). Anatomical details are to be taken from the description of the photographs.

The roentgen picture of the upper lobe root (Fig. 4) also shows many vessel stripes some of which cross and cover each other. The boundaries of the root region can be quite definitely determined and are:

Caudal: The connecting line between the two following points: above the medial border of the Tr. superior (*Tr. s.*), a little lateral from its origin. Below the intersecting point of the lateral border of the R. posterior with the caudal (medial) border of the Tr. intermedius (*Tr. im.*).

Medial: The medial border of the Tr. superior (*Tr. s.*). Then the inner border of the short trunk of the A. apicalis (*A 1*) and its median branch (*A 1a*) to the tertiary branching of the latter.

Lateral: The lateral border of the venous

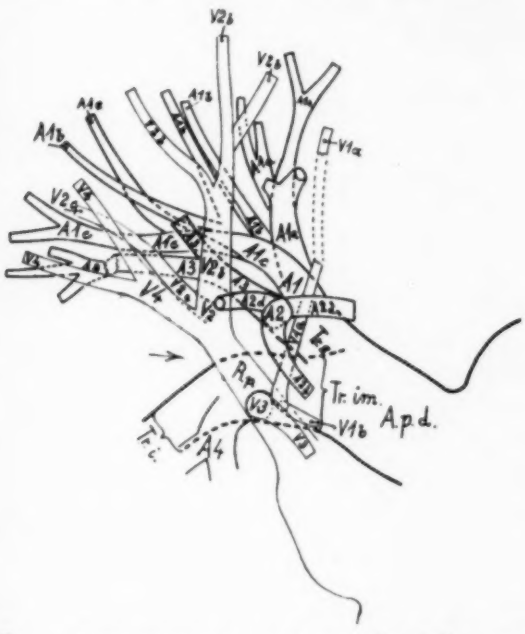

Fig. 4. Roentgenogram tracing of the right upper lobe root. Contours of the arteries drawn somewhat more heavily than those of the veins. →, angle of intersection of the R. posterior and the Truncus intermedius.

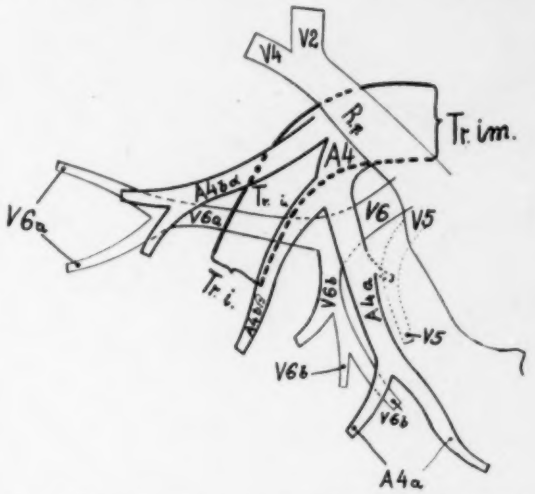

Fig. 5. Roentgenogram tracing of the root of the middle lobe. The V. costalis lobi medii (*V 5*) was not shown in the roentgenogram. Its position is indicated by dotted lines. A clot was found in this vein which had prevented the entrance into it of the contrast medium. Also the first secondary branches of the R. costalis descendens (*A 4b β*) were not definitely recognizable in the roentgenogram. They lie as the anatomical specimen (Fig. 3) shows about at the level of the branch of the vein which runs transversely (*V 6a*). A true R. costalis of the A. lobi medii is not developed here because its two branches (*A 4b α* and *A 4b β*) here originate separately.

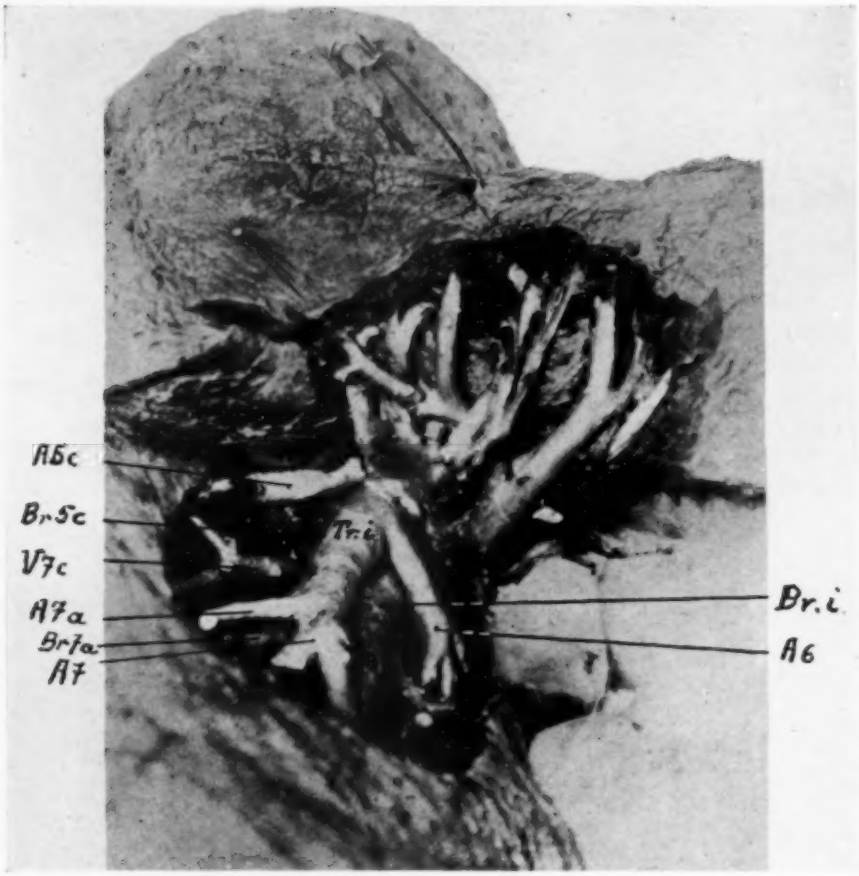

Fig. 6. Root of the right lower lobe, anterior view. The middle lobe is turned upward, lower lobe dissected out from in front. The Tr. inferior of the pulmonary artery (*Tr. i*) visible throughout its length; medially from it the Tr. inferior of the bronchus (*Br. i*). The origin of the A. basilaris anterior (*A 6*) is displaced upwards (variant). As a result of this only one ventral artery, the A. axillobasilaris anterior (*A 7*), passes out from the end of the Tr. inferior. From their branches directed laterally, the R. horizontalis of the apicohorizontal artery and bronchus (*A 5c, Br. 5c*) can be recognized and a R. axillaris of the anterior axillobasilar artery and bronchus (*A 7a, Br. 7a*).

R. posterior (*R. p.*) and the V. axillaris (*V 4*) to the bifurcation of this vein.

Peripheral: A line, straight or slightly convex outward, which connects the point of bifurcation of the V. axillaris (*V 4*) with the tertiary branching of the R. mediastinalis of the apical artery (*A 1a*).

In the mass of the lobe root the vessels and bronchi radiate in all directions to the lobe mantle. Between them, quite far toward the center, lies a typical group of lymph nodes. The vessels of the anterior lower part of the lobe, especially the medial group of branches of the A. apicalis (*A 2a, A 2b*), and often also the R. costo-pectoralis descendens (*A 4d*) and the corresponding veins (*V 1b, V 3*), often take a direction that is predominantly sagittal. Therefore the tendency to an orthoroentgenograde image. In all the other vessels the frontal-parallel direction predominates.

b. The Root of the Middle Lobe (*Fig. 3 and 5*). The cranial part of the root of the middle lobe lies directly against the lower lobe root. Caudally it is separated from the latter by a thin mantle containing air. Its trunks are: the A. lobi medii (*A 4*), the bronchus lobi medii and the two middle lobe veins: the V. paramediastinalis (*V 5*) and the V. costalis lobi medii (*V 6*). The

anatomical location (Fig. 3) and the roentgen conditions (Fig. 5) are quite simple, as the vessels in the sagittal view lie for the most part side by side. The roentgen boundaries are as follows:

Cranial: A line which runs from the origin of the A. lobi medii (*A 4*) in an oblique direction medially and caudad—approximately parallel to the R. posterior (*R. p.*)

This boundary corresponds to the upper border of a cut surface which cuts the trunk of the middle lobe parallel to the interlobar or mediastinal lobe surface. The bronchus lobi medii is synonymous with the first ventral hyparterial bronchus according to Aeby.

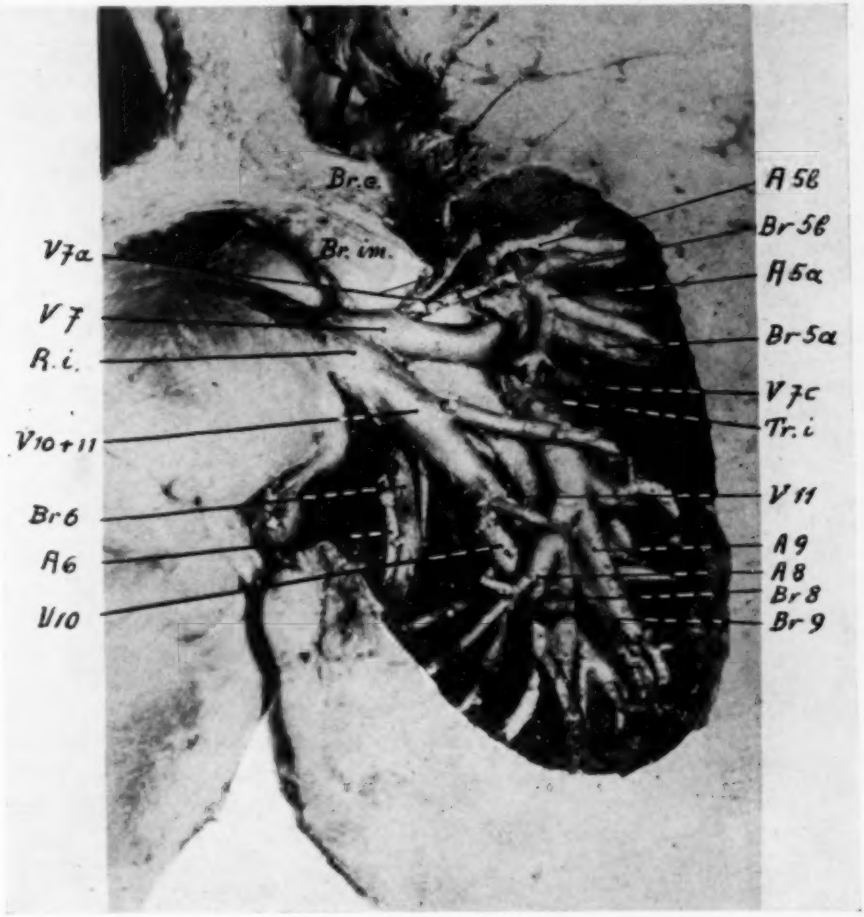

Fig. 7. Root of the right lower lobe from behind. *Description*: The connection with the chief bronchus is preserved so that it and also the Br. epiarterialis (*Br. e*) and the Br. intermedius (*Br. im.*) are visible. The dorsocranial boundary of the lower lobe root runs above and parallel with the V. apico-horizontalis (*V 7*). The trunk of this vein lies free; of its branches the slender R. retrobronchialis (*V 7a*) and the R. horizontalis (*V 7c*) are visible. Of the artery and bronchus of the same name only branches are visible; these are the R. retrobronchialis (*A 5a*, *Br. 5a*) and the R. apicalis (*A 5b*, *Br. 5b*). In the lower region of the lobe root the dorsal branching of the Tr. inferior into two arteries, the A. basilaris post. (*A 8*) and the A. axillobasilaris post. (*A 9*) can be seen. They are accompanied by the bronchi of the same names (*Br. 8* and *Br. 9*). The R. inferior of the lower pulmonary vein (*R. i.*) and its dorsal portion (*V 10* and *11*) as well as its division into the V. basilaris post. (*V 10*) and the V. axillobasilaris post. (*V 11*) can be seen throughout its course. The ventral part of the vein is hidden behind the bronchus; only the place where it is given off from the R. inferior is barely visible. The A. basilaris anterior (*A 6*) and the bronchus of the same name (*Br. 6*) lie deep down and far medially.

Medial: The median border of the V. paramediastinalis (*V 5*) from its entrance into the lobe to its second division.

Lateral: The boundary begins at the outer border of the A. lobi medii. Bending laterally it passes over the R. costalis axillaris (*A 4bα*) at a distance of about 1 cm. from its origin. Then it runs along the lateral border of the R. costalis descendens (*A 4bβ*) to the division of the latter.

Peripheral: The lateral part of this boundary is formed by the lower border of

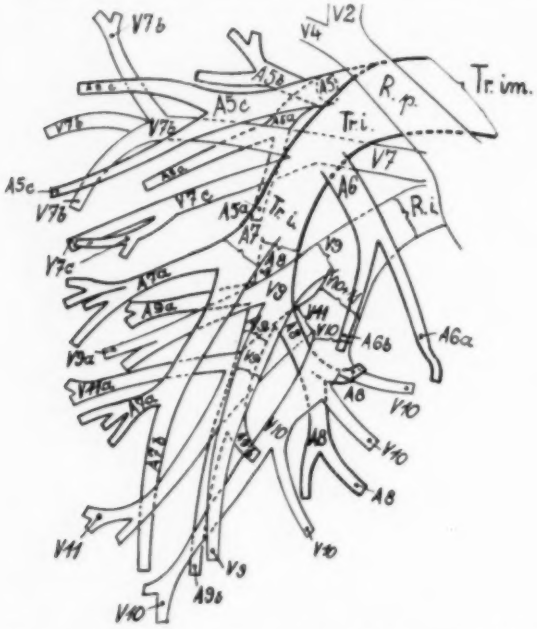

Fig. 8. Roentgenogram tracing of the root of the right lower lobe. The origin of the A. basilaris anterior (*A 6*) displaced upward.

the R. axillaris of the V. costalis (*V 6a*). This line is to be lengthened medially to the giving off of the secondary branch of the V. paramediastinalis (*V 5*).

In the mass of the lobe root a median (paramediastinal) and a lateral (costal) group can be distinguished. The structures of the former take a steep, almost vertical course or often even one curved inward. In the costal group an oblique direction laterally or laterally and downwards predominates. Only the short trunk of the A. lobi medii is often directed sagittally.

c. The Root of the Right Lower Lobe (*Fig. 6, 7 and 8*). The arterial trunk of the lower lobe root is formed by the Tr. inferior of the A. pulmonalis (*Tr. i.*) The lobe bronchus by analogy may be designated as the Tr. inferior of the chief bronchus (*Br. i.* in Fig. 6). The venous trunk is the V. pulmonalis inferior and its branches.

This lobe root is a broad and long complex. The lateral part of it is occupied by the Tr. inferior of the pulmonary artery, dominating in the hilar shadow. The latter, however, has no anatomical predominance —in regard to caliber, length, etc.—over the corresponding bronchus and vein (see Fig. 7). It is therefore incorrect if, in judging the hilar shadow, attention is directed only to the shadow of the artery.

In the lower lobe root an upper and a lower segment can be distinguished. The former is formed by the A. apico-horizontalis (*A 5*) and its branches, as well as the bronchus of the same name (*Br. 5*) and the vein (*V 7*). In the lower segment the terminal division of the Tr. inferior into a ventral and a dorsal portion takes place; the veins pass between these. The anatomical pictures show the details.

In the roentgen delimitation of the lower lobe root the following points are to be considered. The ventral and dorsal borders of the anatomical boundary surfaces do not lie at the same level. The branches directed laterally, especially the branches of the apico-horizontal artery, bronchus and vein, lie close together, partly in the region of the Tr. inferior (*Tr. i.*). The angle at which they are given off is filled with connective tissue and in places also with lymph nodes. Also the angle of intersection of the transverse branches of the vein and bronchus with the Tr. inferior is partly filled with connective tissue. Therefore, the air-containing nuclear lobules in many places cannot immediately touch the lateral aspect of the Tr. inferior, but can only come to within 0.5 to 1 cm. of it. This fact is taken into consideration in the following description of the boundaries.

Cranial: A line directed medially and caudad which, passing over the pulmonary

artery and bronchus, connects the origin of the A. apico-horizontalis (*A 5*) with the mouth of the V. apico-horizontalis (*V 7*).

Medial: The boundary line crosses over the V. apico-horizontalis (*V 7*) and the R. inferior of the lower pulmonary vein (*R. i.*) in the hilar region. Then it follows the median border of the R. inferior and the V. basilaris posterior (*V 10*) to the secondary division of the latter.

Lateral: In the region of the A. and V. apico-horizontalis (*A 5*, *V 7*) the boundary bends outward laterally about 1 cm. Then it runs at a distance of about 0.5 cm. parallel to the external border of the Tr. inferior (*Tr. i.*) and the A. axillo-basilaris anterior (*A 7*). It ends at a secondary branch of this artery 2 to 3 cm. below the bifurcation of the Tr. inferior.

Peripheral: A line straight or slightly convex downward running from the secondary branching of the V. basilaris post. (*V 10*) to the corresponding secondary branching of the A. axillo-basilaris anterior (*A 7a*).

As the basilar vessels often give off several branches of a secondary character, in case of doubt that division point is to be selected which is about 2 to 3 cm. distant from the end of the Tr. inferior.

The mass of the lower lobe root is given a broad solid foundation of shadow by the inferior trunk of the pulmonary artery. But the many branches of vessels and bronchi which cross the trunk dorsally and intensify or clear up the shadow must not be overlooked. The interstices in the root network are filled with abundant connective tissue masses and lymph nodes. The latter are located at many angles of division of the arteries and bronchi, but particularly between the chief trunks of the A. pulmonalis and the bronchus (Engel). The beginning parts of the A. apico-horizontalis (*A 5*) and the R. retrobronchialis (*A 5a*) are often directed almost sagittally backward. In the other vessels and bronchi, the frontal-parallel component of the direction predominates. They run in the upper part of the root horizontally or

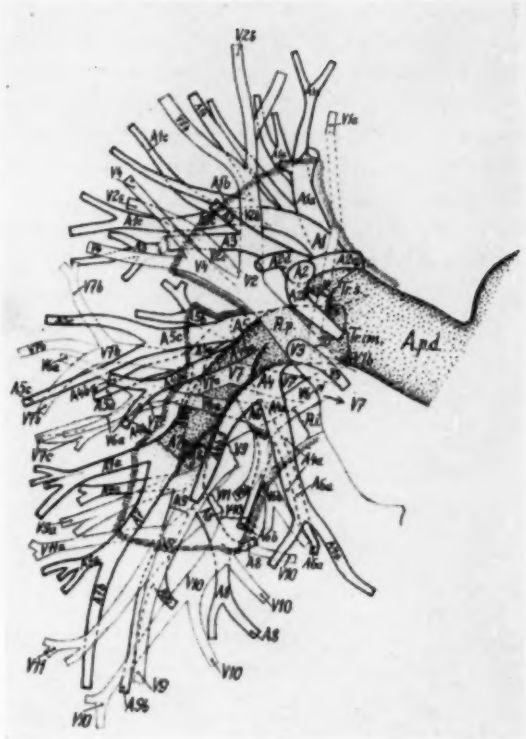

FIG. 9. Identification chart of the pulmonary vessels. Outlines of the arteries black; outlines of the veins grayish black; trunk and branches of the A. pulmonalis dotted lines; outlines of the whole hilum shadow hatched. Corresponding roentgenogram, Figure 1. For method of preparation, see section B.

slightly obliquely upward or downward. In the lower part the arteries and bronchi, analogous to those of the middle lobe, run in a medial group steeply and in a lateral group obliquely downward. The veins show a greater tendency to obliquity because they pass to the corresponding arteries from their mouths which lie farther medially and caudad.

THE HILAR SHADOW AS A WHOLE

The hilar shadow as a whole, as Figure 9 shows, is connected continuously with the vessel shadow. It has, so to speak, to be shelled out from the latter. We find that the hilar shadow is a combined picture of the lobe root and the lung pedicle.

If the identification chart of the lung vessel is considered from the point of view of its lobar composition, the following points

are apparent: The greater part of the upper lobe root and the caudal segment of the lower lobe root are projected free, that is they are not covered by the pictures of other lobe roots. Also by far the greater part of the lung pedicle is overlaid only a little by pictures of the lobe roots. On the other hand, the whole middle lobe root is covered by the upper part of the lower lobe root.

For this reason it is possible to make a

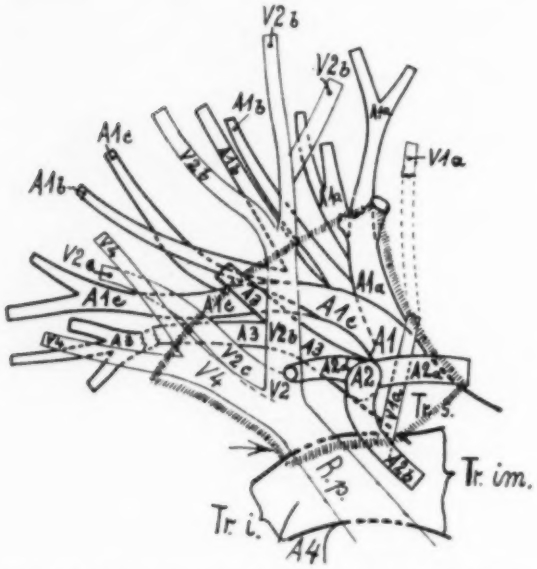

Fig. 10. The "upper end piece" of the hilar shadow. Boundaries hatched; →, angle of intersection between the R. posterior and the Tr. intermedius.

peripheral delimitation of the hilar shadow from the lung markings: take the corresponding boundaries of the lobe roots. And there is also a simple principle for dividing the hilar shadow into the following four sections:

1. The "upper end piece," that is the free part of the upper lobe root.

2. The "pedicle piece," that is the region of the lung pedicle.

3. The "middle piece," that is the projection area common to the middle lobe root and the upper part of the lower lobe root.

4. The "lower end piece," that is the free part of the lower lobe root.

I will first describe the above-named sections of the hilar shadow in some detail. A hatched line in the sketches (Fig. 10 to 13) shows the boundaries of these areas. These sketches are parts taken from Figure 9. They furnish the foundation for a summarizing discussion of the hilar shadow as a projection picture of the complete lung root.

1. *The Upper End Piece of the Hilar Shadow* (Fig. 10). As a rule only a small median-caudad tip of the upper lobe root is projected onto the pedicle piece. Therefore, only the lower boundary of the section in question deviates a little from the caudal boundary of the upper lobe root.

Caudal boundary: The lateral (cranial) shadow border of the Tr. intermedius of the pulmonary artery, continued from the vertex of the angle of its intersection (→) with the R. posterior (*R. p.*) to the point where the Tr. superior (*Tr. s.*) is given off.

Other boundaries: Identical with the boundaries of the upper lobe root previously described.

The structure of the upper end piece is decidedly striped. But the clear zones between the vessel shadows are formed only by the air-columns of the bronchi. For the lobe root is, as mentioned above, a closely wound convolute of tubes containing blood or air, which, to be sure, are surrounded by lobules of lung tissue but do not allow the latter to penetrate between their chief constituents. Among the vessels, the R. posterior of the upper pulmonary vein (*R. p.*) is particularly prominent. Also the V. apicalis (*V 2*) and its R. apico-centralis (*V 2b*) are noticeable because of their steep, often almost vertical course upward. Also the A. apicalis (*A 1*) or its R. mediastinalis (*A 1a*) is frequently distinctly recognizable. Finally attention is to be called to the characteristic angle of intersection (→) of the R. posterior with the Tr. intermedius (*Tr. im.*). This is an important point of orientation to the significance of which I will return later.

Orthoroentgenograde cross sections are found quite frequently. They generally belong to the system of the A. pectoralis (*A 2*), either to the trunk itself or to the

secondary branches, especially the R. sterno-pectoralis horizontalis (*A 2a*) or the R. costopectoralis descendens (*A 2d*). More rarely branches of the other vessels are struck in the orthorentgenograde direction.

Normal lymph nodes are not shown. On the other hand, calcified lymph nodes are quite often found, especially in the region of the branching of the Br. epiarterialis and in the angle of division of the arteries and bronchi of the first order.

2. *The Pedicle Piece* (Fig. 11). This is the massive section of shadow medial-caudad from the upper end piece of the hilar shadow. The more or less extreme outward bending of the Tr. intermedius of the pulmonary artery (*Tr. im.*) takes place in it. Further chief constituents are: the proximal part of the venous R. posterior (*R. p.*), the trunk or the Tr. intermedius of the chief bronchus and the beginning part of the Br. epiarterialis. As the pedicle piece disappears toward the mediastinum in the middle shadow only a partial delimitation is possible.

Cranial boundary: Identical with the caudal boundary of the upper end piece.

Lateral boundary: The lateral border of the R. posterior of the upper pulmonary vein. The caudal and medial boundaries are covered.

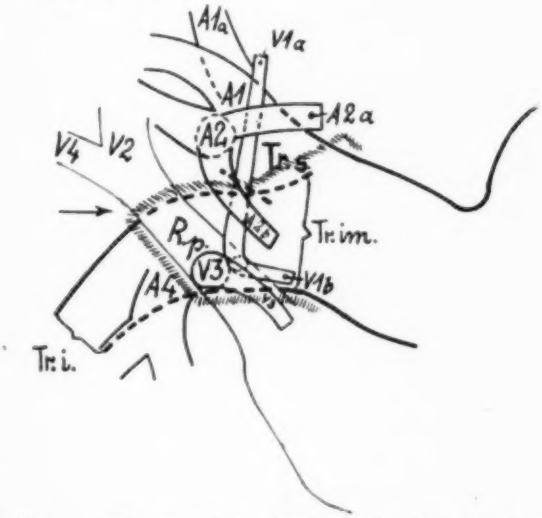

Fig. 11. The "pedicle piece" of the hilar shadow; boundaries hatched.

The pedicle piece shows only little structure. As a rule, the R. posterior (*R. p.*) stands out somewhat in the arterial shadow. The chief bronchus often produces a broad clear band. Also the beginning part of the Br. epiarterialis is not infrequently visible as a clear band running transversely.

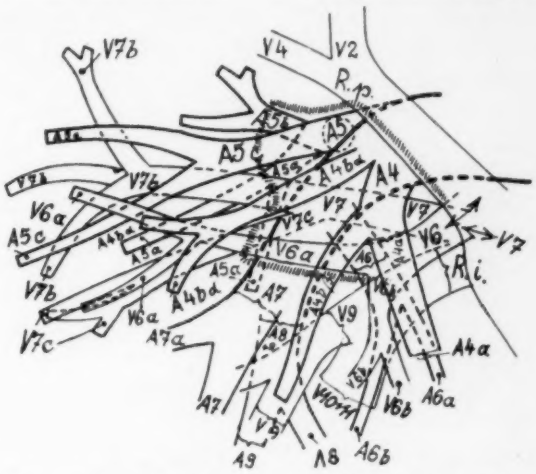

Fig. 12. The "middle piece" of the hilar shadow. Boundaries hatched. The A. basilaris anterior (*A 6*) is projected into this field medially from the Tr. inferior in the region of the clear space of the large bronchial trunk, because its origin is displaced upward (variant).

On the contrary, the upper lobe branches (*A 2b*, *V 3*) projected onto the pedicle piece are only rarely visible in the dense shadow.

Orthoroentgenograde cross sections: Occasionally the V. pectoralis (*V 3*) or the V. subpleuro-mediastinalis can be seen in cross section. Sometimes also the beginning of the A. apico-horizontalis is projected in the orthoroentgenograde direction into the lateral boundary section of the pedicle piece. In general, these findings are quite rare.

The lymph nodes of the pedicle piece belong in part to the tributary lymphatics of the upper lobe, in part to the tributary lymphatics of the middle and lower lobes. They are normally invisible, even when they are quite large. Calcifications are quite frequent; they may occur throughout the pedicle piece and get their lymphatics from any of the lobes of the lung.

3. *The Middle Piece* (Fig. 12). The mid-

dle piece of the hilar shadow lies laterocaudad from the pedicle piece. Its being made up of two root regions makes the picture more complicated and the analysis more difficult. In demarcating it, it must be remembered that the middle lobe root ordinarily appears to be somewhat displaced medially. The lateral marginal region of the middle piece is therefore as a rule made up only of constituents of the lower lobe root.

Cranial boundary: Identical with the caudal boundary of the pedicle piece.

Lateral boundary: Identical with the upper part of the lateral boundary of the lower lobe root.

Medial boundary: Corresponds to the medial boundary of the middle lobe root but is for the most part covered by the heart and mediastinum.

Caudal boundary: This corresponds to the lower boundary of the middle lobe root, that is the R. axillaris of the V. costalis (*V 6a*). Practically it is indicated by the strip of shadow of a vein which crosses the clear space of the chief bronchus and the Tr. inferior of the pulmonary artery transversely. If two such transverse shadows are visible in this region, the lower one is to be taken as the boundary.

Strictly speaking, the R. axillaris costalis (*V 6a*) forms the ventral part of the caudal boundary of the middle piece and the V. apico-horizontalis (*V 7*) the dorsal part. The position of these venous shadows with relation to each other is quite variable. It depends on the anatomical conditions and on the focussing which of them lies further caudal in the picture. The above-named veins also are not infrequently projected over each other. Roentgen differentiation between them is often impossible. But no great error can be made if, in case of doubt, the lower venous stripe is assumed to be the boundary.

The structure of the middle piece acquires a certain massive character from the broad shadow of the Tr. inferior. But, on close examination, quite a rich structure is found, which appears particularly clearly when a suitable technique for making the picture is used. The two transverse venous shadows—the R. axillaris-costalis (*V 6a*) and the V. apico-horizontalis (*V 7*)—have been mentioned before. They are characterized by the fact that they become visible as transverse stripes also medially from the Tr. inferior in the clear space of the large bronchial trunk. In the clear space of the bronchus it is also not infrequently possible to see the shadow stripes of the descending middle lobe vessels (*A 4a*, *A 4bβ*, *V 6b*), running obliquely or vertically, if they are not covered by the mediastinal shadow. The Tr. inferior of the pulmonary artery is crossed, however, not only by the two veins named above, but also by the R. costalis axillaris of the artery (*A 4bα*) and the bronchi belonging to them. The region where the A. apico-horizontalis (*A 5*, *A 5a*, *A 5b*, *A 5c*) branches and the branching of the bronchus of the same name also appear in the laterocranial region of the middle piece. Quite varied pictures appear in vivo because of the varying visibility and combination of these structural details, all of which, however, come within the normal.

Orthoroentgenograde cross sections are sometimes produced in the sagittal picture by the following vessels: the trunk of the A. lobi medii (*A 4*) or the first part of its branches of the first order (R. paramediastinalis and R. costalis), the latter especially when it originates alone; also the beginning of the A. apico-horizontalis (*A 5*), or its R. retrobronchialis (*A 5a*). The cross section shadows are often multiple.

The normally invisible lymph nodes of the middle piece may be located in the middle lobe or the lower lobe. Generally only the lymph nodes lying in the lateral marginal zone belong definitely to the lower lobe. The greatest percentage of calcified lymph nodes is found in the middle piece, among all the localizations in the hilar shadow (see Herrnheiser[3]).

4. *The Lower End Piece of the Hilar Shadow.* The lower end piece is the caudal prolongation of the middle piece and is formed by the distal part of the lower lobe

root. As the latter generally extends obliquely laterally toward the center of the lobe (Fig. 7) the lower end piece does not touch directly on the mediastinum but is surrounded on the median side also by a layer of tissue containing air. For the same reason the distal parts of the middle lobe vessels are not, for the most part, projected onto the lower lobe roots but farther in the median direction. Therefore the boundaries of the lower end piece can be determined quite definitely:

Cranial boundary: Identical with the caudal boundary of the middle piece.

Lateral boundary: It coincides with the lower end of the lateral boundary of the lower lobe root.

Median and caudal boundaries: Identical with the corresponding boundaries of the lower lobe root.

Structure. The anatomical composition of the caudal root end, made up of numerous vessels and bronchi closely connected with each other, is analogous to the structure of the upper lobe root. Therefore in the roentgen picture also there is a structural similarity of the end pieces, which is manifested in the predominance of a striped character. The network of the vessel shadows shows a great variability. In general, the shadow stripes show a tendency to a steep downward course in the median region and to an oblique course in the lateral region. Only the terminal part of the Tr. inferior of the pulmonary artery and the R. inferior of the pulmonary vein give a somewhat broader shadow. The R. inferior, however, is frequently covered by the mediastinal shadow.

Large orthoroentgenograde cross sections are as a rule not seen in the lower end piece of the hilar shadow.

In the caudal part normal lymph nodes are found in much smaller numbers and smaller in size than in the other parts of the lung root. Therefore calcifications are also more rarely seen in the lower end piece of the hilar shadow.

Summarizing Study of the Hilar Shadow (Fig. 9). The total picture of the hilar shadow can easily be reconstructed from the description of its individual sections. The determination of the boundaries (Fig. 9) is contained by implication in our previous reports. I will summarize it only for the sake of completeness.

Cranial: A line which connects the bifurcation of the V. axillaris (*V 4*) with the

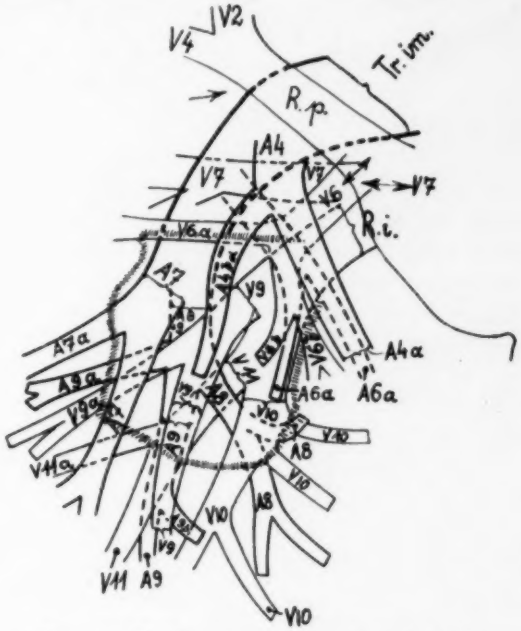

FIG. 13. The "lower end piece" of the hilar shadow. Boundaries hatched.

tertiary branching of the R. mediastinalis of the apical artery (*A 1a*).

Lateral: The lateral border of the V. axillaris and its R. posterior (*R. p.*) to the vertex of its angle of intersection with the arterial Tr. intermedius. Then the boundary bends outward laterally in the region of the origin of the A. apico-horizontalis and runs on parallel to the outer border of the Tr. inferior and the A. axillo-basilaris ant. (*A 7*), at a distance of about 0.5 cm. from them. It ends in the region of the secondary branching of the latter artery about 2 to 3 cm. below the Tr. inferior.

Caudal: The connecting line between the above-named region of the secondary branching of the A. axillo-basilaris anterior and the corresponding secondary branching of the V. basilaris posterior (*V 10*).

Medial: An upper and a lower part of the boundary are to be differentiated from each other. Above: the medial border of the Tr. superior (*Tr. s.*), of the A. apicalis (*A 1*) and of the R. mediastinalis of this artery (*A 1a*). Below: the medial border of the V. basilaris posterior (*V 10*) and of the R. inferior of the lower pulmonary vein.

This boundary determines the size and form of the hilar shadow. Its size seems to be greater, its form plumper and its structure more varied than would appear from Assmann's well-known definition of the hilar shadow. The latter limits his conception essentially to a part of the root of the lung, namely to the larger vessel trunks, particularly the arterial ones. These do, to be sure, give the most intense shadows but are less important as sites of disease. Our conception of the hilar shadow as "the region of projection of the whole root of the lung," on the contrary, includes *all parts* which may be the starting points of pathological processes and may therefore be of clinical importance.

In Figure 9 the trunk and primary branches of the pulmonary artery are marked by dotting. A comparison with the area of the whole lung root, outlined by cross-hatching, shows the differences in form and size resulting from the difference in the two definitions.

It is not possible to give a definite normal size and a definite normal shape of the hilar shadow. There are considerable physiological variations in these respects which are to be attributed to anatomical causes, which, however, can often be demonstrated roentgenologically. The size and shape of the lobes of the lung, differences in the type of branching of the vessels, differences in the position of the root of the lung in the thorax and other things that affect the projection, play a part in it. So *different normal types* of the hilar shadow in living subjects must be established. I cannot go into any further detail in regard to these types within the limits of this article.

As an example, I may mention that there are cases in which the upper lobe is particularly large; in that case the upper end piece of the hilar shadow and often also the pedicle piece dominate at the expense of the other sections. On the contrary, in other cases the upper lobe is small, but the middle lobe highly developed; the middle piece then predominates correspondingly, etc. In cases of the "dendroid" type of branching, the hilar shadow appears compressed, especially at the ends, while in the "thamnoid" (bushy) type of branching it seems more abundantly branched and shows greater structure. Of course the partial covering by the mediastinal shadow must be taken into consideration in judging the size and shape.

The structure of the hilar shadow in the pedicle piece and the middle piece shows chiefly a solid character, in the two end pieces predominantly a striped one. In regard to structural details reference is made to the description of the individual shadow segments. This shows the fact, which is of practical importance, that in the hilar shadow there are a number of striking structures which, as a rule, can also be clearly identified roentgenologically. These leading structures create to a certain extent a frame within which the other details can be arranged. From this point of view, starting with the leading structures, the pictures of different hilar shadows can be compared with each other. From this point of view, too, the identification chart of the lung vessels (Fig. 9) which, strictly speaking, holds true only for a certain specimen and a certain direction of the rays, can be used, *mutatis mutandis*, as a basis of comparison.

Among the above-mentioned leading structures are the following:

(a) The trunk and branches of the pulmonary artery, to which Assmann has called attention.

(b) The R. posterior of the upper pulmonary vein. Its position in the curvature of the A. pulmonalis and its oblique course over the bronchus and trunk of the artery laterally and upward are characteristic of it.

(c) The angle of intersection of the R. posterior of the vein and the Tr. intermedialis of the pulmonary artery. It is important because the interlobar fissure between the upper and middle lobes ends at its vertex (see Fig. 3). The position of the angle in the hilar shadow, therefore, gives a certain measure of the proportionate size of the two lobes. Moreover, the boundaries of the upper end piece, the pedicle piece and the middle piece come together at its vertex.

Vital roentgenograms in which the interlobar fissure is represented as a hairline show that this line always leads to the vertex of the angle of intersection. Even when only a part of the interlobar hairline is visible when the latter is properly prolonged it leads directly to the angle.

(d) The R. inferior of the lower pulmonary vein and the V. basilaris posterior. This shadow is characterized by its origin medially from the chief bronchus and by its oblique course, in contrast with the steep descent of the artery.

(e) The V. apicalis and the R. apicocentralis. An often almost vertical course from above downward to the R. posterior and its position in the lateral area of the upper end piece is typical of this vein shadow.

(f) The transverse vein at the boundary between the middle piece and the lower end piece. Details in regard to this are discussed in section 3.

Orthoroentgenograde cross sections of vessels have two preferred localizations in the hilar shadow: the lower part of the upper end piece and the boundary zone between the pedicle piece and the middle piece.

With reference to the regions to which the lymph node shadows belong the following rules can be established:

In the upper end piece they belong exclusively to the upper lobe.

In the pedicle piece they may drain from any lobe.

In the middle piece the lymph nodes in general may belong either to the middle lobe or to the lower lobe. Only in the lateral marginal region lymph nodes are to be attributed only to the lower lobe.

In the lower end piece they drain only the lower lobe.

It has been the object of our roentgen-anatomical examinations to create a foundation for the objective judgment of the vital hilar shadow. The question of whether and to what extent this object has been attained would require such a detailed discussion that I cannot enter into it here. I may only say that this question has been answered in the affirmative by my experience in this respect. A further discussion of the conditions in the living subject must be reserved for a special work.

SUMMARY

1. The roentgen anatomy of the hilar shadow is the roentgen anatomy of the root of the lung. This may be divided into the lobe roots and the lung pedicle. The outer boundary of the root of the lung—according to Felix's representation of the lobe structure—is to be located at the peripheral border of the "inner zone of the lobe nucleus."

2. After a discussion of the technique of examination, the anatomy of the different lobe roots of the right lung is discussed on the basis of anatomical specimens. Roentgenogram tracings show the sagittal projection figure of the lobe roots, their boundaries and their structure.

3. From the lobar composition of the lung root a classification of the hilar shadow into the following four sections is derived: (*a*) the "upper end piece," that is, the larger part of the upper lobe root that is projected free; (*b*) the "pedicle piece," that is, the region of the lung pedicle; (*c*) the "middle piece," that is, the common area of projection of the middle lobe root and the upper part of the lower lobe root; (*d*) the "lower end piece," that is, the free caudal part of the lower lobe root. These sections are described separately and their demarcation from each other, their structure, the projection position of orthoroent-

genograde vessels cross sections and calcified lymph nodes, etc., taken into consideration.

4. The definition of the hilar shadow as a projection of the whole lung root includes a larger region than Assmann's well-known definition. There are different normal types of the hilar shadow. The structure of the hilar shadow shows several striking "leading structures" which make orientation easier and create a basis for comparison. From the position of calcified lymph nodes in the hilar shadow, conclusions can be drawn to a certain extent in regard to the region where they belong.

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THE RESPIRATORY FUNCTION OF THE DIGESTIVE TRACT AS THE BASIS OF ROENTGENOGRAPHIC LIFE TEST*

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THE problem of air being present in the digestive tract is an old one. It has been known for a long time that every human stomach contains some air, and it has also been known since the work of Breslau that air appears in the stomach of a newborn infant simultaneously with his first extra-uterine breath. The mechanism of the entrance of air into the stomach still remains uncertain, and the fate of air which has found access into the stomach is unknown.

In 1891 Bouveret supposed, on the basis of his observations of three hysterical

FIG. 1. Case 1. A.P., aged eighteen. Suffers from "loud eructation." Incessantly belches air. On an empty stomach a great bladder.

women suffering from so-called "loud eructation," that the air eructated by these invalids entered the stomach or the esophagus by swallowing; then the air being pushed back by the antiperistaltic movements of the esophagus put into vibration the soft palate bringing about the loud sound. Bouveret called this "aérophagie hystérique." Many succeeding authors have maintained and developed this con-

FIG. 2. Case 1. Immediately after a contrast meal. The stomach bladder has not increased at all.

clusion; they have explained every accumulation of air in the stomach as "aérophagie"—i.e. swallowing of air. There are, however, many facts which disagree with this conclusion of the mechanism of air entering the stomach. First, it must be observed that in order to swallow a small bubble of air strong peristaltic movements of the

* From the Roentgenological Institute (Director, Prof. J. G. Dillon) of the Moscow District Clinical Institute (Director, Dr. A. S. Awanessow). Presented at the Fifth International Congress of Radiology, Chicago, Ill., Sept. 13–17, 1937.

FIG. 3. Case II. E.N., aged twenty-three. Suffers periodically from "loud eructation" with a swelling of the epigastrium. Esophagus during inspiration. Expanded esophagus, entrance of the stomach and stomach bladder are clearly seen.

esophagus are necessary, while Barclay proved in 1930 that there are no peristaltic movements, at least in the upper part of the esophagus up to the height of the clavicle. Second, swallowing air is, of course, possible and roentgenologists observe it sometimes, but this is only possible if something else is swallowed, even saliva, as there can be no empty swallowing. Third, according to my own observations of those classical invalids with "loud eructation," it could clearly be seen that the stomach air bubble before feeding was larger than after it, i.e. no air entered the stomach during the swallowing of the contrast mass (Fig. 1 and 2).

Hence, if air does not enter the stomach by gulping it can get there in only one way, namely, suction during inspiration. This was mentioned by Leven, then by Schultz; and Oser maintains that the air enters the stomach by way of aspiration. Magnusson by an experiment on himself proved that air enters the stomach by inspiration.

In order that the air may enter the esophagus on inspiration, a negative pressure in the esophagus during inspiration is, however, necessary which actually must take place with any inspiratory extension of the thorax, this being proved by manometric measurements of Schreiber (1883) as well as by esophagoscopic observations of Mickulicz, Gottstein, and others (an obvious opening of the esophagus during inspiration, and nearly a complete collapse of the walls during expiration). This opening of the lumen of the esophagus may be seen at roentgen observation if the thorax of the observed person presents favorable natural contrasts (Fig. 3 and 4).

Further entry of air into the stomach during inspiration necessitates a negative pressure in the stomach, which is also proved by measurements of Schreiber as

FIG. 4. Case II. Moment of expiration. Esophagus noticeably contracted. Entrance of the stomach and stomach bladder are not seen.

well as those of Hildebrand and Hess. However, for the entrance of air into the stomach at the moment of inspiration it is necessary that besides a negative pressure in it there must also be a free passage outside of gulping; the latter being possible only when the cardiac sphincter is always open, or if it does not exist at all. The presence of the cardiac sphincter has not been proved by anatomists, and nerve control is not known to anyone. Its existence is entirely denied by a number of authors. Thus Hacker affirms that the muscular sphincter between the esophagus and the stomach lies in the *esophageal opening of the diaphragm.* This is also maintained by Gottstein as well as Rosenheim. Mickulicz states that in his esophageal investigations he never observed any sphincter-like closing between the esophagus and the stomach and the passage between them was always open. Bayer denies the existence of a closing

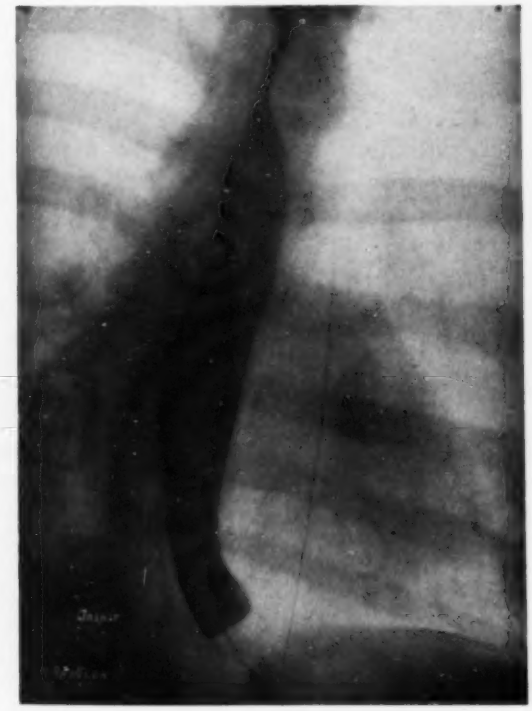


Fig. 5. Case III. M.J., aged thirty-seven. Indeterminate complaints of neurasthenic character. Moment of inspiration. Contrast meal found obstruction in the esophagus. An entire obstruction of the diaphragmatic part of the esophagus.

Fig. 6. Case III. Moment of expiration. Contrast meal passes freely into the stomach.

sphincter in the cardia and he states that the passage of food masses into the stomach is controlled by respiratory movements between separate gulps. Guns' observations on the act of swallowing made him suppose that the contraction of the entrance of the stomach is formed by the diaphragm—the cardia by itself presents no hindrance to masses of food. Finally Caballero, on the basis of his experiments, affirms that the cardia is always open and presents no sphincter. The food is retained at the entrance of the stomach not by the cardia but by the epicardia. Only *periodical respiratory movements of the diaphragm* allow the food, under ordinary conditions, to pass into the stomach.

On the basis of a great number of careful personal roentgen observations I can affirm that during the act of swallowing the food under normal conditions passes into the stomach, depending only on the contractility of the diaphragm and the respiratory

function of the diaphragmatic sphincter of the esophagus; the phase of inspiration—a systolic phase of the diaphragm and its loop-like sphincter in the esophageal hiatus causes an obstruction of food above the cardia (Fig. 5), and the phase of expiration —a diastolic phase of the diaphragmatic sphincter—allows a free passage of the food through the cardia into the stomach (Fig.

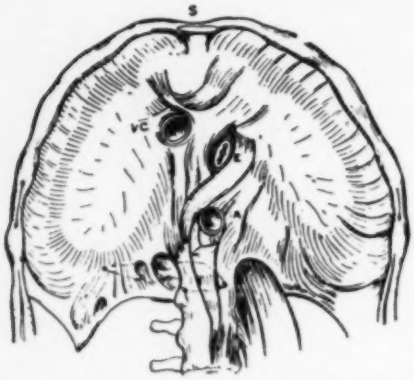


FIG. 7. The under surface of the diaphragm. What the author (Jackson) has called the "diaphragmatic pinchcock" is shown at *E*. The muscular and tendinous prolongations of the diaphragm surround the esophagus so as to form a typical pinchcock. This pinchcock normally closes the esophagus and opens it at the proper moment in the deglutitory cycle (from Chevalier Jackson).

6). At a regular inspiration this phenomenon as a law can be observed in everyone. By this mechanism of ingress of the food-lump into the stomach at the moment of diastolic (expiration) weakening of the diaphragm is explained the fact that to drink at one draught a large quantity of liquid is only possible at the moment of *expiration*, and immediately after the drink a deep inspiration follows which is the deeper the larger the drink. Hence in accordance with many authors such as Strauss, Guns, and others, the condition known as "cardiospasm" ought to be called "phrenospasm." Chevalier Jackson thinks that the so-called cardiospasm represents always an insufficient opening of the only place of constriction of the esophagus—diaphragmatic sphincter—and calls it "diaphragmatic pinchcock" (Fig. 7). And actually even in persons who are seriously ill such a constriction of the esophagus is found in the diaphragmatic part (Fig. 8).

The air which enters the esophagus during inspiration, can freely enter the stomach as well as leave it, depending only on the respiratory function of the diaphragm. It is quite evident that in case of a disturbance in the function of the diaphragm we must have also some changes in the air content of the stomach which take place in all cases without exception. This was observed by Franck, and I, in a great number of cases of paralysis of the diaphragm, showed that a large accumulation of air in the stomach was one of the most certain symptoms of a paralysis of the diaphragm (Fig. 9). This accumulation of air is explained by a bending of the diaphragmatic end of the esophagus because of the high position of the diaphragm, thanks to which the air easily enters the stomach but leaves it with difficulty. When the function of the diaphragm is restored, as happened in one of my cases, the normal air content of the stomach is at once restored (Fig. 10, 11 and 12).

Besides these indirect proofs a number of direct proofs of the entrance of air into the stomach by means of the repiratory act can be mentioned. Schindler says that by

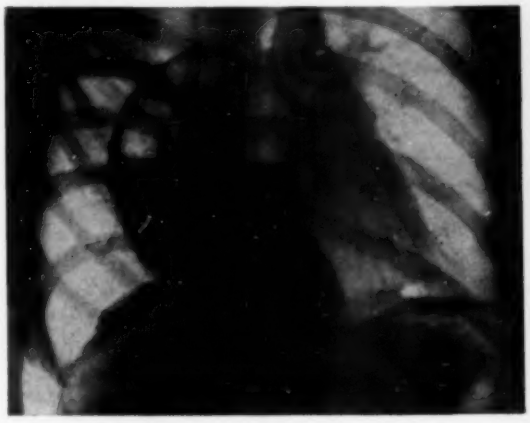


FIG. 8. Case IV. J.R., aged twenty-six. Four years of dysphagia. Serious case of a so-called cardiospasm. It is easily seen that the contraction takes place in the diaphragmatic part of the esophagus and not in the cardia.

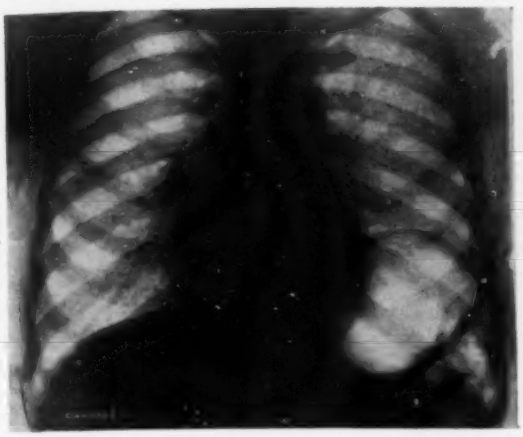


Fig. 9. Case v. A.S., aged forty-two. A blind bullet wound in the chest with affection of the left phrenic nerve. Paralysis of the left diaphragm. A large accumulation of air in the stomach.

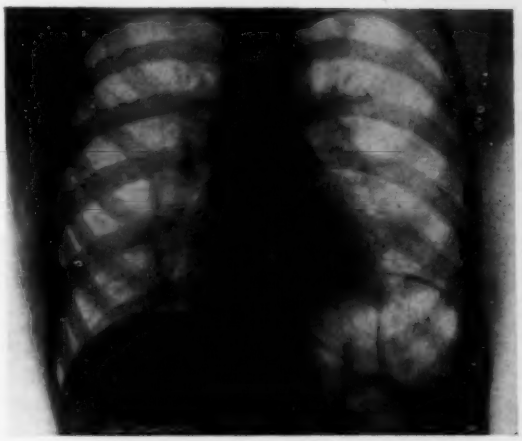


Fig. 11. Case vi eight days later. A sudden paralysis of the left diaphragm as a result of an infectious neuritis of left phrenic nerve. A high position of the left diaphragm. Large accumulation of air in the stomach bladder.

gastroscopy we can see how the stomach expands during inspiration and how it contracts during expiration. In a case of congenital atresia of the esophagus, if only its lower end is connected by a fistula with the trachea, the air gets into the stomach and the intestines in spite of the utter impossibility of any swallowing (Fig. 13, 14 and 15) (Vogt, Sussman). Ventriloquists, as is known, use the stomach as an aerial reservoir filled with air by inspiration and emptied during speech. Invalids whose larynx has been extirpated because of malignant neoplasm learn to use this manner of speech (Voorhoeve, Kharshak).

Now, it is clear that the air gets into the stomach by the act of respiration and its stay in the stomach depends on the function of the respiratory muscles. Thus it follows that with an established respiration we have as an inevitable result air in the stomach. Pathologic-anatomic postmortem examinations, as well as roentgenographic

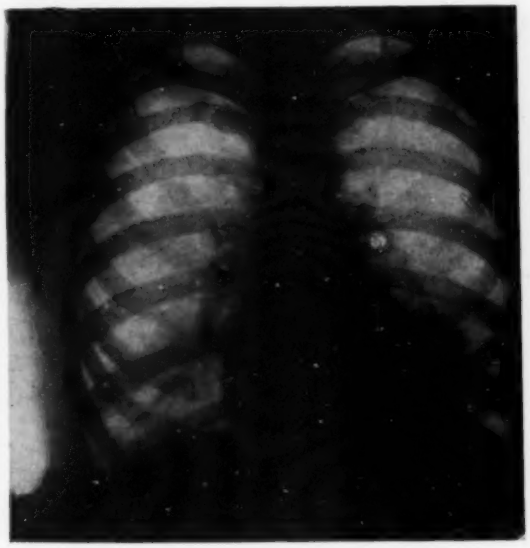


Fig. 10. Case vi. M.Kr., aged twenty-four. A case of paratyphus. Regular function of the diaphragm. A nearly complete absence of stomach bladder.

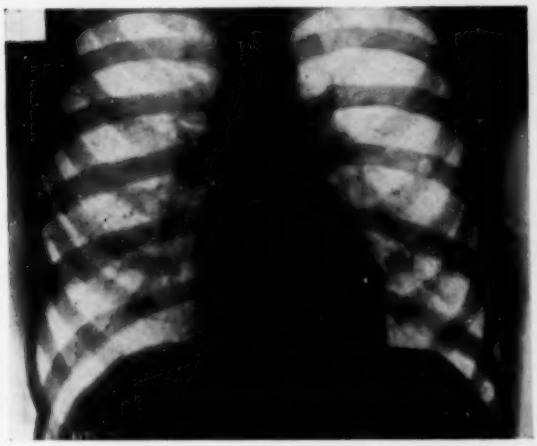
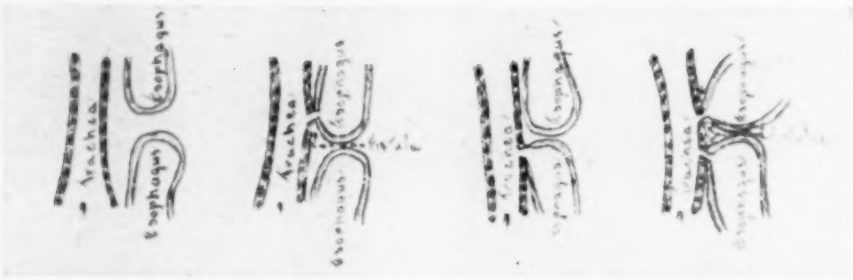


Fig. 12. Case vi ten days after Figure 11. A complete recovery of the function of the diaphragm. Hardly noticeable quantity of air in the stomach.

studies, prove that until the moment of appearance of extra-uterine respiration there are no traces of air in the digestive tract. The abdominal cavity as well as the thorax of a stillborn shows on a roentgenogram always a uniform density, only the outlines of the skeleton being clearly seen, while the organs of the abdominal cavity as well as those of the thorax are absolutely impossible to distinguish from each other. The roentgen image of the fetus imcapable of living or prematurely born differs from that of a full-term newborn only by the character of the development of the skeleton (Fig. 16 and 17). Quite another picture characterizes infants born alive and who have breathed for even a few minutes. Depending upon how much one or both of



Type 2 Type 3(*a*) Type 3(*b*) Type 3(*c*)

FIG. 13. Types of a congenital atresia of the esophagus. Type 3(*b*) of special interest (from Vogt).

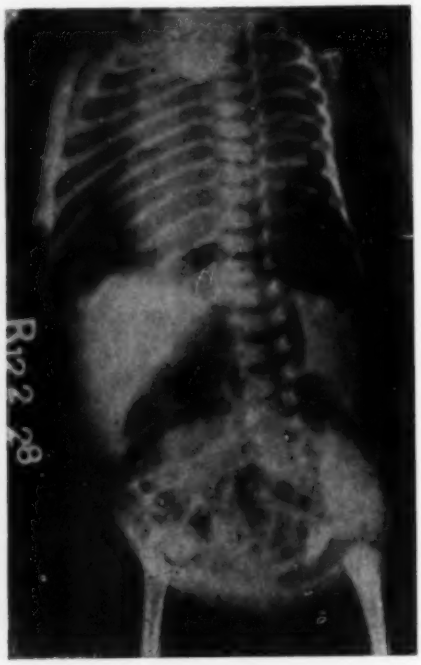


FIG. 14. Type 3(*b*) atresia showing complete obstruction of the upper esophageal segment. Air in the stomach and intestines denotes a connection between the lower segment and the trachea or bronchus (from Vogt).

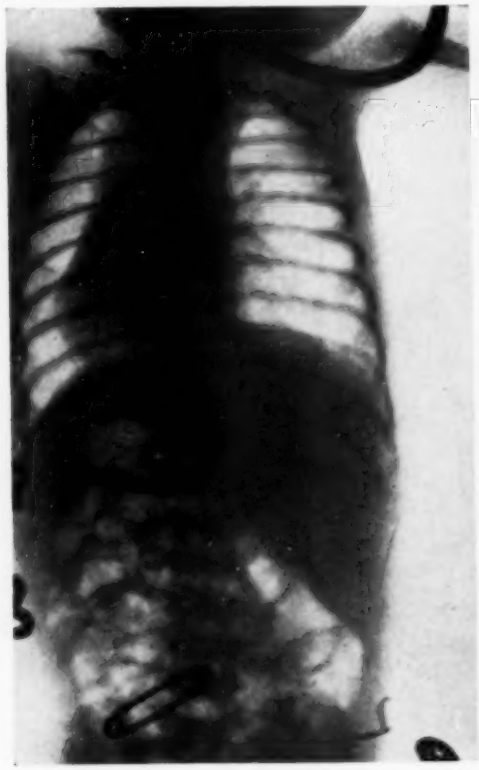


FIG. 15. Congenital atresia of esophagus. Contrast meal in the upper esophageal segment. Air in the stomach and intestines.

their lungs are expanded, the outlines of the heart and the bronchial tree are more or less differentiated. But the most important and substantial difference concerns the abdominal cavity. If the infant makes even a few respiratory movements and if the lumen of the esophagus is not blocked by any

Fig. 16. Case vii. A stillborn fetus of twenty-eight weeks. Uniform gray picture of the thorax and abdominal cavity. Heart and lungs cannot be distinguished from each other. No air in the abdominal cavity.

extraneous body or mucus a certain amount of air is to be found in the stomach or intestines, and the deeper and longer the respiratory movements of the thorax, the larger the amount of air (Fig. 18 and 19). We can often observe that at the first moments of life the stomach is so full of air

Fig. 18. Case ix. Tk. A live full-term infant. Lived five minutes. The lungs are still unexpanded and the heart and airless lungs cannot be distinguished from each other or from the abdominal viscera. A small quantity of air in the stomach and in the duodenum.

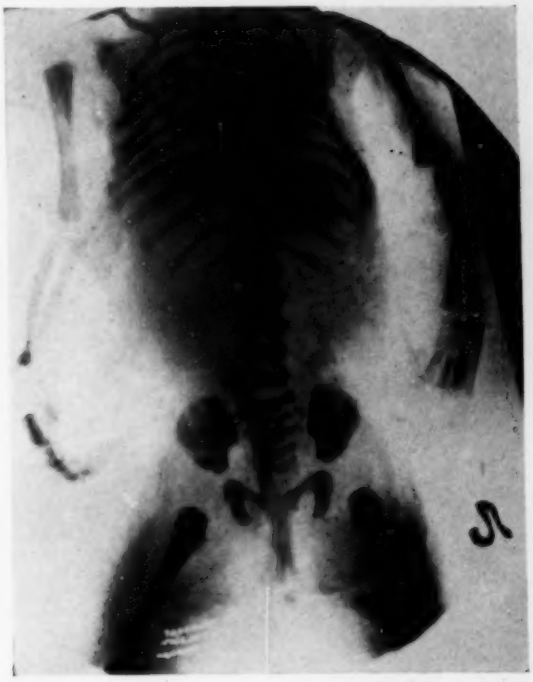


Fig. 17. Case viii. Cher. A stillborn full-term fetus. The same uniform gray picture of the thorax and abdominal cavity. No traces of air.

it expands to maximum size. Sometimes rather energetic peristaltic waves are to be clearly seen (Fig. 20). Attention must be

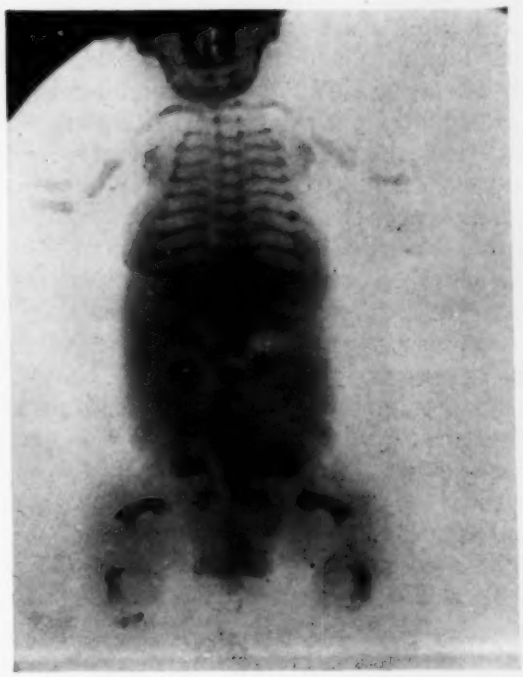


FIG. 19. Case x. Tit. Twenty weeks' fetus born alive. Lived seven minutes. A complete atelectasis of the lungs. Clearly noticeable air in the stomach and duodenum.

called to the fact that the air inspirated into the stomach spreads so fast that in a few moments the large intestine may be already filled with air (Fig. 21).

It has not yet been possible to establish exactly a correlation between the degree of the expanding of the lungs and that of the air-filling the stomach and the intestines, but it can be supposed that the more air in the stomach, the less the lungs expand. In any case the finding of air in the stomach of the newborn after the first extra-uterine breath is so absolute, and at the same time roentgenographically so well established, that roentgenography must be acknowledged to be the most reliable means of determining whether an infant was born alive or was stillborn. It must also be counted as the most sensitive means of finding air in the stomach. Our investigations show that a quantity of air (0.2 sq. cm.) which cannot be ascertained by the so-called hydrostatic gastric test can be easily detected by roentgenography.

Now it remains only to see what significance can be attached to the air inspired into the stomach. Undoubtedly the fate of the air which has found access to the stomach and the intestines does not differ from that of any other gases in the digestive tract, i.e. it can be freely absorbed into the blood. This is especially true of oxygen which can be dissolved in any liquid of the digestive tract and as such be absorbed by the blood in the same way as it infiltrates into the alveoli of the lungs. There is also no anatomical impediment to such an absorption of oxygen through the walls of the digestive tract, as the digestive tract embryologically comes from the same anlage as the respiratory tract. Comparative physiology presents indisputable proofs of an absorption of oxygen into the digestive tract of some vertebrates. Thus the swimming bladder of fishes can under certain conditions assist respiration. There

FIG. 20. Case xi. Ogn. A newborn full-term infant. Lived ten hours. Lungs are not being completely expanded. The stomach is distended by the air present.

are fishes which besides branchial respiration have intestinal respiration, and the river groundling, *Cobitis fossilis*, breathes in such a way: it emerges from the water and gulps air, which passes through the intestines and comes out of the anus.

There are also direct proofs of the possibility of using the digestive tract of a human organism as a respiratory organ. Kratter states that the infants upon whom postmortem examinations were made by him and other physicians, and who lived, according to medical records, for several hours, showed no air in their lungs, but some air was found in the digestive tract. Although Kratter does not mention any possible use of the air contained in the digestive tract for gaseous exchange, nevertheless it is quite clear that if the air entered the stomach and not the lungs the oxygen feeding could take place only through the digestive tract.

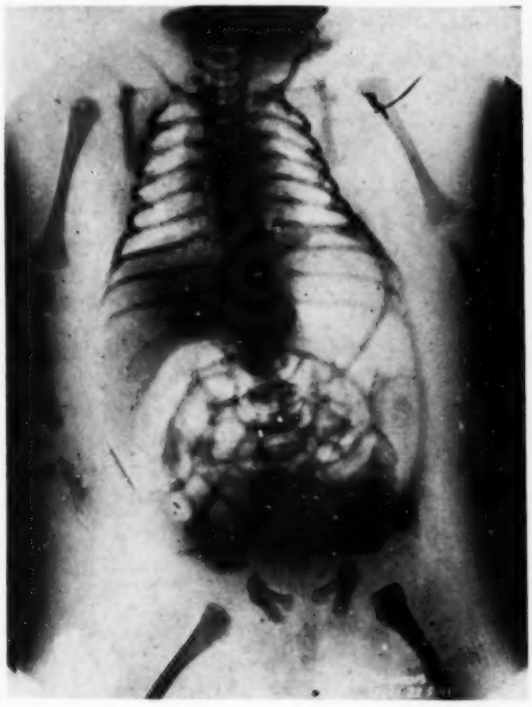


Fig. 21. Case XII. Jouck. A newborn full-term infant. Lived twenty minutes. Lungs are well expanded. The stomach is extremely distended by air. A considerable quantity of air is present in the intestines.

Fig. 22. Case XIII. Str. A newborn full-term infant. Lived twenty-five minutes. Atelectasis of the lungs. A considerable quantity of air in the thin intestines.

Similar cases were observed in our practice. Case I, an infant who, according to records of a Lying-in Hospital, was born alive and lived twenty-five minutes, was examined roentgenologically shortly after its death. On the roentgenogram it was quite impossible to distinguish the heart from the lungs, which indicated their atelectatic state. At the same time the stomach and intestines contained air (Fig. 22). The anatomist who made the postmortem examination found a congenital atelectasis of the lungs (Minutes of the Pathological-Anatomic Institute, Jan. 14, 1935, N40). It is quite evident that this infant who lived twenty-five minutes could breathe only by the stomach; air could not get into the lungs owing to their complete atelectasis.

Case II is of still greater interest. The infant, according to records of the Lying-in Hospital, was a full-term newborn. His

FIG. 23. Case XIV. Brnd. A newborn full-term infant. Lived five hours. Cried, but feebly. Complete atelectasis of the lungs. A large quantity of air in the stomach and intestines.

birth cries were feeble. There was no need of any means to enliven it. But it lived only five hours. The roentgenogram taken soon after death showed a complete atelectasis of both lungs, and at the same time a great quantity of air was found in the stomach and intestines (Fig. 23). Thus, on the basis of this roentgenogram we come to the conclusion that the infant could not breath with its lungs. The autopsy (Minutes of the Pathologic-Anatomic Institute, April 22, 1935, N352) fully confirmed our conclusions. The lungs did not expand at all and only in the large bronchi could their lumina be detected. All the sections of the lungs, even those of the upper part taken for investigation, sank in water and were composed of liver-like tissue. No other inference can be drawn from this fact than that the infant did not breath with its lungs at all and that it could live and even cry only by means of the air which was in its digestive tract and which could get there only by means of respiratory movements of its thorax.

Thus the gastrointestinal respiration of man is well established, but nevertheless I cannot state definitely what rôle it plays in the gaseous balance of adults in their normal state. It is possible that it has some significance in some pathological cases when the respiration of the lungs is affected. But as to the newborn whose lungs expand completely only at the end of the first week, there is no doubt that the gastrointestinal respiration is a regular subsidiary gaseous exchange which begins to function during any disturbance or stoppage of the respiration of the lungs. From this point of view the presence of air in the digestive tract of a newborn is proof of its respiration, and therefore the roentgenographic test of life proposed by me possesses the force of a medicolegal witness to show whether an infant was born dead or alive.

CONCLUSIONS

1. The only place of obstruction of the lower end of the esophagus is the loop-like para-esophageal muscle of the diaphragm functioning synchronously with the respiratory movements of the diaphragm.

2. The term "aerophagy" which does not correspond to the mechanism of accumulation of air in the stomach should be replaced by a more correct term, namely that of "gastrospiry."

3. The air enters the stomach and intestines of the newborn and adults during the inspiratory movements of the thorax. The air which can be swallowed has no substantial significance either in adults or in infants.

4. At present there is sufficient evidence to consider it well established that as well as pulmonary respiration every human being has a gastric respiration, the latter being subsidiary but in some cases it may become prevailing.

5. The presence of air in the digestive tract of a newborn can serve as proof of extra-uterine respiration and hence as proof that the infant was born alive.

6. The roentgen examination of a stillborn fetus at any time of its uterine life

never reveals any traces of air in the digestive tract.

7. Correctly made roentgenograms of dead fetuses that were breathing even for a very short time always disclose the presence of air in the stomach or intestines, no matter how small its quantity may be. Therefore such a roentgenogram serves as a test of life.

8. A roentgenographic test of life does not prevent further investigation of any kind.

9. A roentgenographic test of life is the most reliable and sensitive of all such available tests.

10. A roentgenographic test of life makes it possible to have a permanent document possessing the force of court decision.

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LAMINAGRAPHY OF THE SPHENOID BONE*

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ROUTINE roentgen examinations of the sphenoid bone permit study of the superior and posterior surfaces of its body and the sphenoidal air sinuses. The usual projections employed are the lateral, the coronomental, the submento-occipital, and the occipito-oral projection through the open mouth. The greater part of the sphenoid bone, however, is hidden by the superjacent osseous shadows. These obscure the anterior and inferior surfaces of the sphenoid bone body and the alae which form part of the orbits and the anterior and middle cranial fossae, regions particularly difficult to study either on single or stereoroentgenographic films.

The applicability of laminagraphy to skull roentgenography has been mentioned previously.[2,3,6] This technique, which has been aptly termed "body section roentgenography," permits examination of any predetermined plane within a structure by eliminating the images of the regions above and beneath that plane.[4,5] Thus, by making a series of lateral cuts an inch apart sectional roentgenograms of the skull visualizing the sphenoid bone in layers are obtained. Coronal cuts can also be made, and are useful for examining the antra and ethmoidal sinuses. Special emphasis will be placed on the use of laminagraphy in the examination of the sphenoid bone in this article. Five cases have been selected to illustrate the advantages of supplementing routine roentgenographic procedures with laminagrams. The apparatus used was that devised by Alexander.[1]

A brief description of the anatomy of the sphenoid bone might be helpful before proceeding with the case reports. The body of the bone is roughly cubical in shape and contains two large cavities which are separated by a septum. On its superior surface is the ethmoidal spine, which articulates with the cribriform plate of the ethmoid. Behind this are the chiasmatic groove, the tuberculum sellae, the sella turcica, the posterior clinoid processes, the dorsum sellae and the clivus. The anterior clinoid processes are formed by the medial ends of the posterior borders of the lesser wings. The posterior surface of the body is quadrilateral in form, and is joined during early adult life to the basilar part of the occipital bone. Both lateral walls are united with the great wings and the medial pterygoid plates. The carotid groove lies above the attachment of each wing. The anterior surface presents a midline crest which articulates with the perpendicular plate of the ethmoid. On either side of the crest is an irregular opening of variable size which leads into the corresponding air sinuses. The lateral margins of the anterior surface are serrate and articulate with the lamina papyracea of the ethmoid, completing the posterior ethmoidal cells.

The inferior surface is a solid plate of bone which presents a triangular crest in its midline called the sphenoidal rostrum. This is continuous with the sphenoidal crest on the anterior surface, and is received in a deep fissure between the alae of the vomer. On either side of the rostrum is a projecting lamina, the vaginal process, directed medialward from the base of the medial pterygoid plate.

The superior or cerebral surfaces of the great wings form part of the middle cranial fossae, while their anterior surfaces constitute the posterior portions of the lateral orbital walls. The inferior rims of the superior orbital fissures are formed by the medial margins of the orbital surfaces of the great wings.

The lesser wings are joined to the body of the sphenoid by two roots, the upper one thin and flat, the lower thick and triangular. The optic foramina lie between the two roots. The superior surfaces support part of

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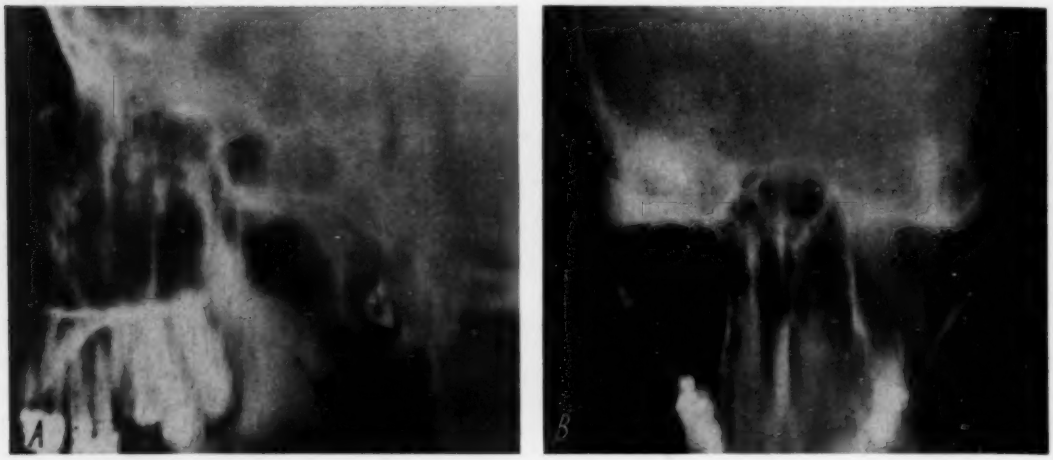

FIG. 1. *A*, laminagram through the mid-sagittal plane of a normal skull illustrating the appearance of the anterior and inferior walls of the sphenoid, the clivus and the basilar portion of the occipital bone. *B*, coronal laminagram through the same skull illustrating the lateral walls of the sphenoid and the sphenoidal air sinus.

the frontal lobes of the brain, the inferior surfaces form the upper boundaries of the superior orbital fissures and the rear of the orbital roofs. Thus the upper surfaces of the lesser wings form the posterior portions of the anterior cranial fossae, while the posterior free borders form the anterior margins of the middle fossae.

The pterygoid processes descend perpendicularly from the roots of the great wings adjacent to their insertion into the body of the sphenoid, and divide into a lateral and mesial plate. These are difficult to identify roentgenologically.

The foramina ovale, lacerum and spinosum can be identified on films of the base of the skull. The foramina rotundum cannot be demonstrated roentgenologically because of their anterior projection into the pterygomaxillary fossae.

REPORT OF CASES

CASE 1. J. K., a boy, aged six, was admitted because of headache, vomiting and moderately elevated temperature. Two weeks before admission he developed sudden paralysis of the right external rectus muscle and twitching of the right cheek. One week later bilateral paralysis of the fourth and sixth cranial nerves appeared, followed by ptosis of the right eyelid.

Right lateral stereoroentgenograms, posteroanterior and anteroposterior views of the skull taken the day of admission revealed normal density of the anterior wall of the sella and the anterior clinoid processes. The posterior wall of the sella and the posterior clinoid processes could not be seen. There was no evidence of a destructive process despite the apparent anomaly which some observers believed might be congenital. The orbital fissures were normal.

An encephalogram done the following day showed normal lateral ventricles. There was a

FIG. 2. Case 1. *A*, right lateral projection of skull, encephalogram. Note the absence of the posterior clinoid processes and the dorsum sellae. The basilar portion of the occipital bone is obscured by the temporal bone shadows. The arrows indicate the soft tissue mass impinging on the air-filled third ventricle.

B, laminagram through mid-sagittal plane. The dorsum sellae, the posterior clinoid are not visible. A destructive lesion involving the basilar portion of the occipital bone is demonstrable.

C, right lateral projection of skull taken one month later. It is about the same as the first study (*A*).

D, laminagram through mid-sagittal plane. The extensive destruction of the entire sphenoid is evident. The arrow indicates soft tissue shadow of the tumor in the oral pharynx.

E, laminagram through the left great wing of the sphenoid. The bone is intact.

F, laminagram through the right great wing of the sphenoid. Note the punctate appearance and the thickening of the bone (arrows). *E* and *F* were taken on the patient's first admission.

A
B
C
D
E
F

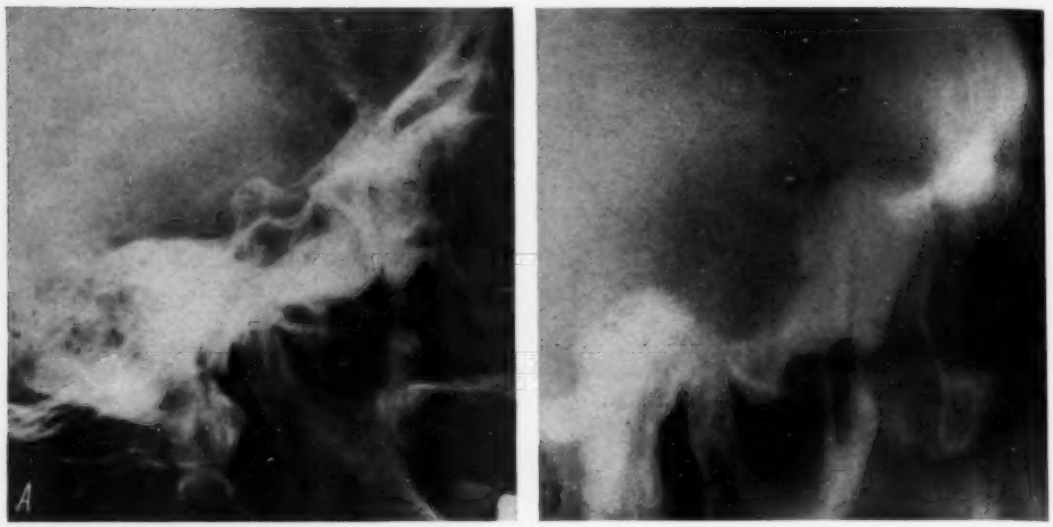

Fig. 3. Case II. *A*, right lateral projection of skull. The sella appears to be normal. The middle cranial fossae are obscured by overlying bone. *B*, laminagram through the right middle and anterior cranial fossae demonstrating the advanced hyperostosis of the lesser and greater wing of the sphenoid. A similar study through the left middle and anterior cranial fossae was normal.

slight defect in the anterior aspect of the third ventricle suspicious of a subjacent tumor mass.

Serial laminagrams were taken two days later. These revealed a definite punctate destructive lesion in the right great wing of the sphenoid with thickening of the bone. A similar laminagram of the left great wing taken for comparison was normal. A section through the midline of the skull showed the dorsum sellae, the clivus and the proximal half of the basilar process of the occipital bone to be destroyed.

The patient was given deep roentgen therapy with some symptomatic relief. He was discharged three weeks later with a clinical diagnosis of probable intrapontine tumor.

One month later he was readmitted because of increasingly severe headaches. Routine roentgenographic examination of the skull showed no definite changes in the lateral projection. In the posteroanterior and anteroposterior views there was slight irregularity of the right superior orbital fissure as compared with the left.

Serial laminagrams revealed extensive destruction of the entire sphenoid bone. The soft tissue tumor mass could be seen projecting into the oral pharynx.

The patient's condition became rapidly worse and he died soon thereafter. Autopsy revealed an extremely large sarcoma involving the base of the skull, the pituitary gland, the nasopharynx and the trigeminal nerves. The entire sphenoid bone was eroded and destroyed by tumor.

Comment. The first definite indications of a bone destroying intracranial neoplasm were obtained by means of laminagraphy. The routine studies did not indicate the pathology since it was obscured by the overlying normal bone shadows. Comparison of the routine views of the skull taken on the first and second admissions showed slight changes. The extent of the destructive process was far better portrayed by comparing the laminagrams made on each admission.

Case II. S. E., male, aged fifty-two, complained of weakness of the left side of his body for about twenty-seven years following a gunshot wound. He had been able to lead a fairly active life. About one year ago he noticed that his right eye lachrymated excessively when exposed to the cold. A sense of fullness and pressure behind the right eye developed and about six months ago it became prominent.

Physical examination revealed weakness of the left upper and lower extremities with gross diminution of muscle bulk. There were increased deep tendon reflexes, sustained ankle clonus and sluggish abdominal reflexes on the

left side. The right eye was proptosed.

Lateral stereoroentgenograms of the skull showed the sella turcica to be normal. Increased density over the posterior ethmoidal cells obscuring the anterior portion of the body of the sphenoid bone was present. On the posteroanterior and anteroposterior films there was increased density of the lateral inferior wall of the right orbit. The orbital fissure on the right side was somewhat narrowed.

Serial laminagrams of the skull showed advanced hyperostosis of the posterior margin of the anterior fossa and the anterior two-thirds of the middle cranial fossa on the right side. A calcified tumor mass was visualized projecting above the posterior aspect of the superior surface of the lesser wing of the sphenoid, invading this region as well as the adjacent middle fossa. The sella turcica was normal. A posteroanterior laminagram 2 inches from the face showed a large calcific mass springing from the lesser wing of the sphenoid. This was egg-shaped, and measured 5 cm. in its long diameter and 3 cm. in its widest portion.

The preoperative diagnosis was right sphemoid wing meningioma with hyperostosis. At operation the hyperostotic region was reached after turning a bone flap low down toward the temporal region. The hyperostosis was then attacked with perforator and large rongeurs and removed piecemeal until most of the bone projecting into the orbit was removed. The removal of the bone was continued until most had been removed, although in the opinion of the operator some undoubtedly remained on the base of the skull. Histopathologic study revealed meningioma with hyperostosis.

Comment. The extent of the lesion could not be estimated from the routine roentgenograms. Laminagrams were essential in this instance to outline the osseous involvment of the anterior and middle cranial fossae by the meningioma.

Case III. J. S., male, aged fifty-six, complained of numbness and anesthesia of the right side of his face from the mandible to the midscalp region and from his nose to the tragus of the right ear for two years. He lost the sense of taste in the right side of his mouth and the right anterior portion of his tongue about one year before admission. Paralysis of the right eyelid which progressed to complete ptosis started one year ago. Six months later his right eyelid gradually lifted because of progressive ocular bulging. Vision in his right eye became blurred and he was blind for six weeks before admission. Hearing in the right ear became progressively worse. There were no headaches, nausea, vomiting or tinnitus. He had occasional transient episodes of vertigo, and salivated from the right side of his mouth without being aware of it.

The patient was euphoric and very talkative. There was a right dacrocystitis and conjunctivitis. The second, third, fourth, fifth, sixth, seventh and eighth cranial nerves were paralyzed and there was weakness of the right twelfth nerve. Proptosis of the right eye was present. The left knee jerk was hyperactive. Diminished plantar flexor responses were elicited on the left side. The optic discs were pale. The spinal fluid pressure was 150 mm. of water; the manometric responses were normal.

Routine lateral stereoroentgenograms, posteroanterior and anteroposterior views of the skull showed normal osseous markings. Serial laminagraphic sections revealed definite thickening of the anterior and inferior aspects of the body of the sphenoid. This was considered indicative of invasion with tumor tissue.

At operation a grayish tumor was seen extending into the orbital plate of the right frontal bone and backwards over the posterior border of the lesser wing of the sphenoid, disappearing into the middle cranial fossa. The tumor, removed piecemeal, was hard, rubbery and adherent to the base of the skull but not to the overlying brain. It was noted that the posterior border of the lesser wing was soft and involved in the pathologic process. Further removal of the tumor revealed that the right optic nerve and internal carotid artery were buried in tumor tissue.

Microscopic study revealed metastatic carcinoma. The primary focus was not discovered. The patient made a good postoperative recovery.

Comment. The laminagrams indicated a metastatic lesion in the anterior and inferior walls of the sphenoid which was not demonstrable on the routine studies.

Case IV. E. L., male, aged fifty-six, became progressively more oblivious of his family for six months before admission. His family noted a change in personality from an alert, aggressive individual to a quiet, meek person who sat

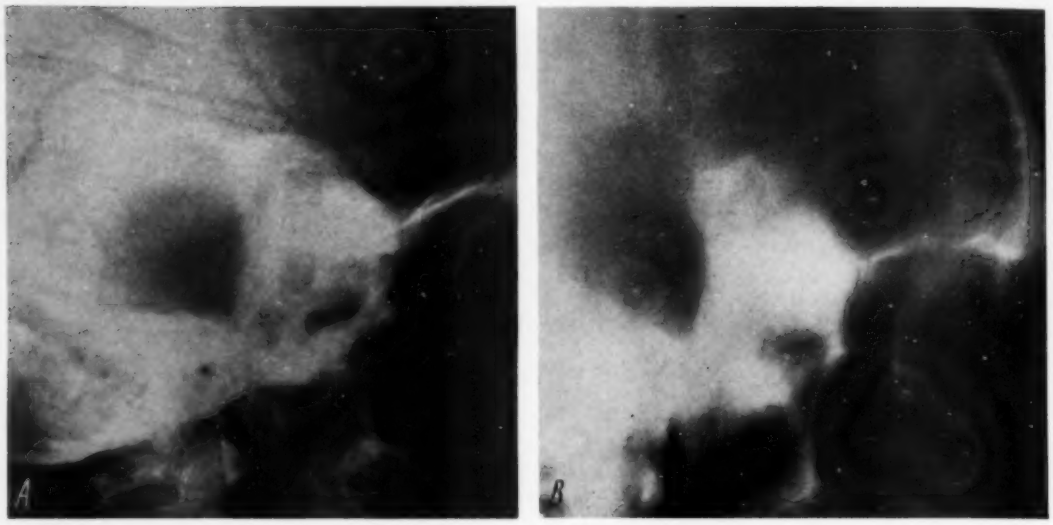

FIG. 4. Case IV. *A*, right lateral roentgenogram showing calcification obscuring the sella turcica. *B*, laminagram through the right anterior and middle cranial fossae. The dense calcific core impinging on the lesser and anterior portion of the greater sphenoidal wing is demonstrable. Involvement of the adjacent portions of the middle fossa with destruction of the cerebral surface of the great wing is present.

quietly by himself. He developed difficulty in cerebration and became slovenly in his habits. There were no symptoms of headache, vomiting or failing vision.

Examination showed loss of memory and personality alterations. Some atrophy of the left optic disc and slight blurring of the right disc were noted. His deep reflexes were slightly increased in the right upper limb. The remainder of the examination was negative.

Routine lateral stereoroentgenograms revealed a calcific triangular shaped mass whose base obscured the sella turcica and clivus. In the posteroanterior and anteroposterior projections this was seen to be limited to the right upper aspects of the orbit, obliterating the superior orbital fissure.

Serial laminagrams showed the sella to be normal. The sphenoid bone body and its air spaces were normal. The clivus was intact. A large calcific mass invading the right lesser and greater wings with a dense core about 3 cm. in diameter impinging on the superior aspect of the lesser wing was demonstrable in sections taken 1 and 2 inches from the right side.

The bone of the middle cranial fossa was invaded by tumor tissue resulting in destruction of the cerebral surface of the right great wing.

The preoperative diagnosis was right temporal meningioma invading the sphenoid bone. At operation considerable erosion of the greater wing of the sphenoid was found where the old calcareous portion of the tumor had been attached. A large part of the tumor including the calcific mass was removed. Microscopic examination revealed a psammomatous meningioma.

Comment. The laminagrams in this instance were helpful in outlining the extent

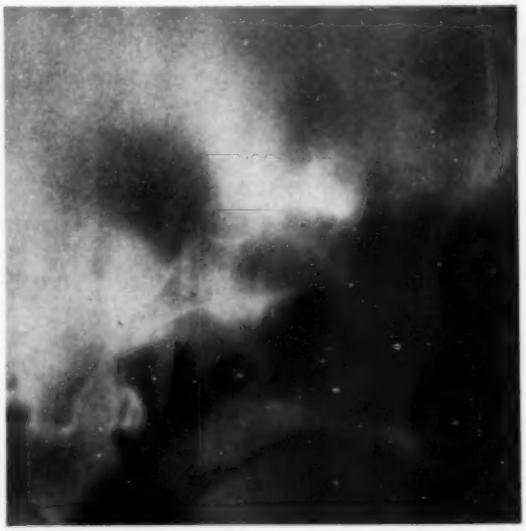

FIG. 5. Case V. Laminagram through the mid-sagittal plane demonstrating metastatic involvement of the inferior wall of the sphenoid bone. This was not demonstrable on the routine studies.

of the osseous involvement of the middle and anterior cranial fossae. The invasion of the greater wing of the sphenoid was particularly well portrayed on the laminagrams.

Case v. R. D., female, aged fifty-nine, was admitted because of continuous nosebleeds for three weeks before admission. The bleeding was most profuse from the right side of the nose. The patient complained of dull pain in the left side of the abdomen for about six months. Gross hematuria occurred for the first time the day before admission.

Examination showed a polypoid mass in the right nares which bled readily when touched. A mass was palpated in the left upper quadrant.

Biopsy of the nasal mass revealed metastatic carcinoma. Intravenous urography showed evidence of a left renal tumor.

Routine study of the sinuses revealed an opacity over the right antrum and nasal passages. There was destruction of the mesial wall of the right antrum. The right ethmoidal cells were cloudy. Lateral views of the skull showed the sphenoid sinuses and the sella turcica to be within normal limits. A base view of the skull showed opacity over the right sphenoidal cells.

Serial laminagrams made in the lateral and coronal projections showed destruction of the anterior and inferior wall of the body of the sphenoid bone. The alae of the sphenoid were intact. Coronal sections revealed involvement of the left as well as the right ethmoid sinuses. The destructive lesion in the antral walls was clearly delineated.

Comment. The laminagrams were helpful in identifying the destruction of the anterior and inferior walls of the body of the sphenoid. This could not be seen on the routine studies. The extent of the involvement of the antra and ethmoids also was better seen on the laminagrams.

SUMMARY

Laminagraphy is useful in the investigation of the sphenoid bone. It is possible to demonstrate the anterior and inferior walls of the body of the sphenoid as well as the individual alae. The middle and anterior cranial fossae can best be studied on laminagrams.

Five cases in which laminagrams supplemented the routine roentgenograms of the sphenoid are reported.

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A SUBSTITUTE FOR THE FATTY MEAL IN CHOLECYSTOGRAPHY*

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CHOLECYSTOGRAPHY is the roentgen study of the gallbladder after the oral or intravenous administration of a suitable opaque substance and it has proved a most valuable diagnostic tool in the hands of the roentgenologist. Although the details of the method vary considerably in different clinics, the routine roentgen studies are best carried out in two stages. In the first portion of the examination, a dose of the opaque medium is administered to the patient and, after the lapse of the proper interval, roentgenograms are made of the gallbladder region. The second portion of the cholecystographic study consists of having the patient ingest a large amount of fat-rich food, waiting one-half hour or longer, and then exposing another set of roentgenograms. However, there are numerous objections to the use of the fatty meal and the purpose of this communication is to describe a means of eliminating these objectionable features.

The meal is used because it is known that fats in the duodenum cause the gallbladder to contract and empty itself either partially or completely. In one hour the normal viscus usually decreases to one-half or less its original size. The foods most commonly used to accomplish this purpose are cream, eggs, and butter, as these are among the most efficient substances in producing emptying of the gallbladder. The amounts and proportions of these foods vary widely in different clinics. An average meal comprises one-half glass of cream, two eggs, butter, and bread or toast. The meal, in addition to indicating the ability of the gallbladder to contract, serves other important functions. It may show stones which because of their density or size are not demonstrable in the large, well-filled gallbladder, as calculi of these varieties are sometimes seen only after the gallbladder has contracted and partially emptied itself. Polyps, adhesions, bands and other pathologic conditions may be demonstrable only during the second portion of the cholecystographic study. In dyskinesia of the gallbladder, the viscus enlarges after the ingestion of the fat-rich foods.

However, as stated above, there are objections to the routine use of the fatty meal. It causes some individuals to suffer from bloating, nausea, and vomiting. Persons who are accustomed to partaking of little or no breakfast frequently rebel at being required to ingest so large an amount of food in the morning when the test is ordinarily carried out. Many patients have an aversion for eggs, cream, or butter.

From the standpoint of the roentgenologist, likewise, the fatty meal is frequently objectionable. If it becomes necessary or desirable to perform roentgen studies of the gastrointestinal tract simultaneously with cholecystography, the presence of food in the stomach requires a wait of several hours and in some instances an entire day for the stomach to empty itself before the barium meal studies can be carried out. It may at times be desired to follow the cholecystographic examination by barium enema studies. The presence of large amounts of food in the stomach not infrequently results in distress, nausea, and vomiting when the colon is filled with the opaque enema, or otherwise interferes with the examination of the large bowel.

Of the foods which are known to produce contraction and emptying of the gallbladder, egg-yolk and lecithin have been found among the most satisfactory. In the endeavor to find a suitable means of overcom-

* The lecithin-egg-yolk mixtures used in this study were supplied through the courtesy of the National Synthetic Co., New York, New York.

ing the above-described objectionable features of the fatty meal, we have carried out a series of observations with concentrated mixtures of these substances. The combination which has given satisfactory results consisted of 30 cc. of a mixture containing 45 per cent egg-yolk and 10 per cent lecithin. This has now been administered to 140 patients. In every instance there has been a good response with contraction or complete emptying of the gallbladder. In no case has there been any complaint of distaste for the mixture or unpleasant reaction after its ingestion.

In about one-fourth of the cases, the stomach has been examined roentgenologically with the opaque meal immediately after the exposure of the films in the second portion of the cholecystographic study, which in our clinic are made one or two hours subsequent to the ingestion of the egg-yolk and lecithin mixture. The study of the barium-filled stomach has not been interfered with in any of these patients and the visualization of the stomach, duodenum, and intestines was not affected. In numerous instances barium enema studies were carried out directly after cholecystography. The studies of the colon were satisfactory and no unpleasant reactions occurred.

Patients in whom only cholecystographic studies were being carried out with no intention of doing either gastrointestinal or colon studies immediately afterwards were given the choice of a fatty meal or the egg-yolk-lecithin mixture. More than half of our patients expressed an aversion to fat-rich foods and chose the other alternative. Some individuals were given a combination of the two or were allowed to supplement the lecithin-egg-yolk mixture with foods of their own selection. This did not interfere with the cholecystographic studies and has given better results than the routine use of the fatty meal.

In our clinic, the usual procedure at present is to give the patient 30 cc. of the egg-yolk and lecithin combination diluted with 30 to 50 cc. of water. If the stomach or colon is to be examined immediately afterward nothing else is permitted. Otherwise, the patient may add foods of his own choice, although we indicate that our preference is for moderate amounts of easily digestible foods. In the hospital, the patient may be kept in the roentgen department or returned to his bed in the interval between the two portions of the cholecystographic study according to the exigencies of the particular case and the convenience of the clinic. For the roentgenologist engaged in private practice the egg-yolk-lecithin with vanilla or other suitable flavoring material may be kept in individual sealed bottles ready to use on dilution with an equal amount of water. This saves the patient the necessity of dressing and going to a restaurant. The use of the above mixture has the advantage of simplicity, economy, and uniformity. It is time saving and has proved more satisfactory than the various fatty meals previously in use.

SUMMARY

In cholecystography, a meal rich in fats is usually used to produce contraction and emptying of the gallbladder.

The fatty meal may in some instances prove objectionable both to the patient and from the standpoint of the roentgenologist.

A mixture containing 45 per cent egg-yolk and 10 per cent lecithin has proved an entirely satisfactory substitute for the fatty meal.

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ROENTGENOGRAPHIC SOFT TISSUE STUDY IN AN ORTHOPEDIC HOSPITAL

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ALTHOUGH the writings and teachings of Carty[1,2,3,4] have emphasized the importance of showing and studying soft tissues roentgenologically, and although Ferguson[5,6] has recorded the vital importance of soft tissue shadows in diagnosis, common medical practice is still hampered by insufficient consideration of these structures.

For many years now at the Hospital for Ruptured and Crippled, where one might think the principal roentgenologic interest would be centered on bone rather than soft tissues, the emphasis has been on soft tissue study. We have found that most roentgen diagnoses are based wholly or in part on soft tissue findings. In principle, we have been in complete accord with Carty and Ferguson; but in method we have differed from the former, who studies soft structures by means of special films, taken with special technique. In our opinion, special films are not necessary for this study. We believe that, in most portions of the body, bones and soft tissues can be studied satisfactorily on the same films, provided that (1) the films are not made unnecessarily dark, and (2) different intensities of illumination are used in studying the films.

TABLE I

CONDITIONS IN WHICH SOFT TISSUE STUDY REPEATEDLY AIDS DIAGNOSIS

Arthritis:	Differentiation of various types, such as acute infectious, rheumatoid, tuberculous.
Bursitis:	Subacromial, radiohumeral, olecranon, trochanteric, prepatellar, Achilles.
Cysts:	Popliteal; of lateral meniscus of knee; of infrapatellar fat pad.
Ganglia:	Especially about wrist.
Neoplasms:	Soft tissue, intracapsular (mostly in knees) and extracapsular.
Tenosynovitis:	Of supraspinatus, of interossei, of tendons lateral to lower extremity of radius.
Trauma:	Sprain; syrovitis and overlying bursitis differentiated; tear of tendons, such as triceps, quadriceps, Achilles; early march foot, often manifested only by a small fleck of soft callus; foreign bodies such as glass and pencil point, intracapsular and extracapsular.
Veins:	Varicosities; phlebitis; angiomas. Evidence that hypertrophy of an extremity was due to local vascular conditions, such as arteriovenous aneurysm, hemangioma, or lymphangioma.

(1) Opinion and taste differ widely as to the ideal density of roentgenograms. The roentgenologic staff at this hospital prefers films of somewhat lesser density than many we see coming from other laboratories, believing that in routine cases increased density gives no further information on bone, but does bring the undesirable consequence of blotting out the soft tissue shadows. We feel that the use of dense films should be limited to special cases where bone producing or sclerotic processes are present, such as old osteomyelitis, Paget's disease, or osteoplastic types of osteogenic sarcoma.

(2) Should there be, however, on the staff of the hospital, or in the clientele of the roentgenologist, surgeons who have a preference for denser films, a compromise can be reached which will satisfy surgeon and roentgenologist. Techniques can be so adjusted that the films are dense enough to satisfy the surgical eye, while at the same time the roentgenologist can indulge his taste for studying soft tissues by using illumination sufficiently intense to visualize these soft tissues. As part of our regular equipment, alongside our viewing boxes and within easy reach, we have a gooseneck lamp, with metal parabola shade with white reflecting surface, equipped with a Mazda 100 watt bulb. With this fixture soft tissues

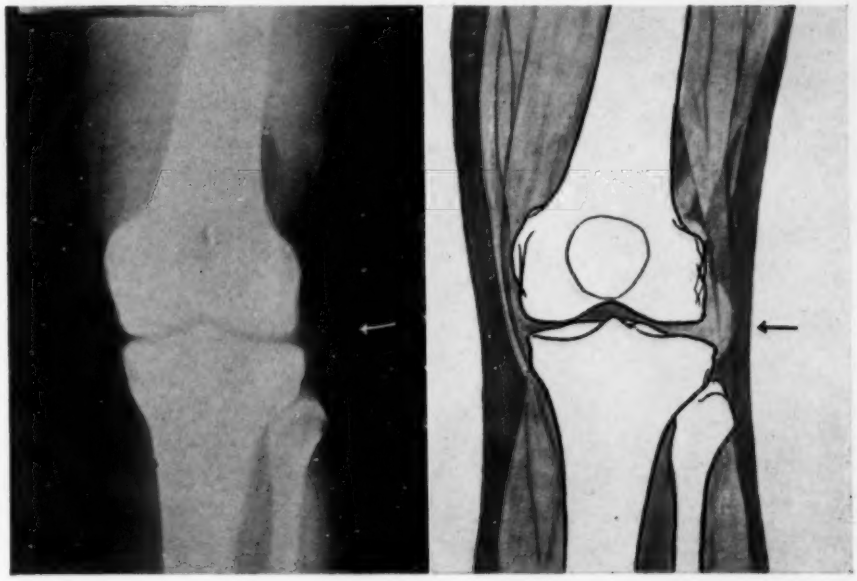

Fig. 1. Cyst of lateral meniscus. Characterized by localized, smoothly rounded soft tissue bulge at lateral margin of the joint.

can be seen clearly in all except films of extreme density.

Brief mention and a few illustrations of conditions in which soft tissue study repeatedly aids diagnosis in an orthopedic hospital may increase the general appreciation of its value. Table 1, by no means all inclusive, lists some of these contributions.

As Ferguson has stated, without soft tissue shadows the diagnosis of arthritis, and especially the differential diagnosis of the various types, is almost impossible. With proper films it is frequently possible to identify acute infectious arthritis, rheumatoid arthritis, and tuberculosis.

Soft tissue films show clearly faint de-

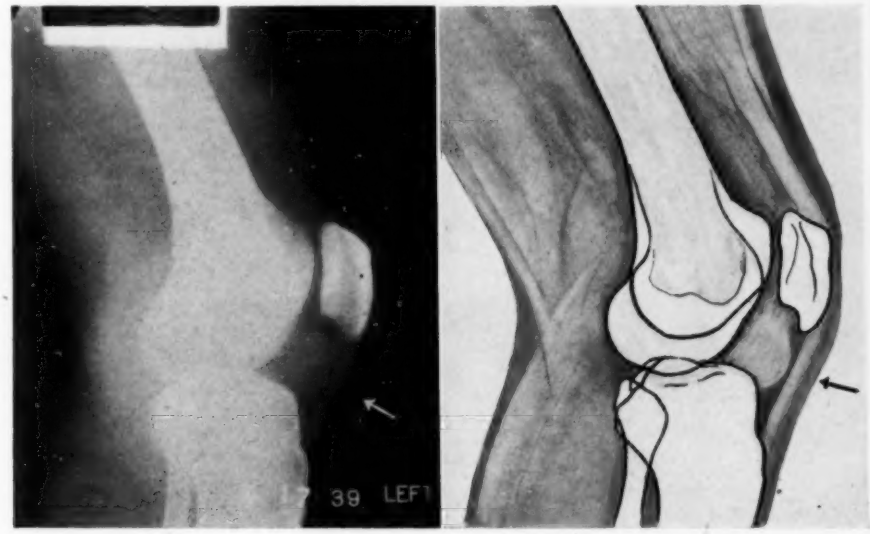

Fig. 2 Cyst of infrapatellar fat pad. Recognized in preoperative roentgenograms only as an unidentified tumor mass.

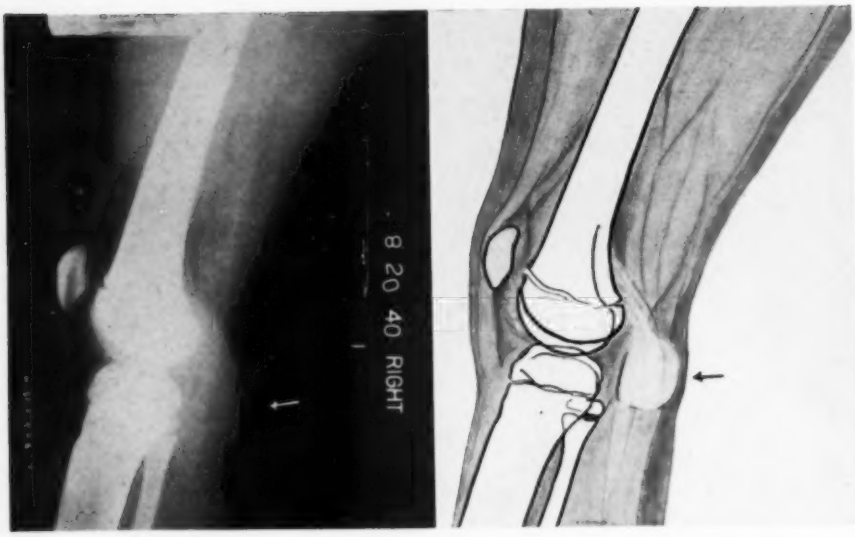

Fig. 3. Popliteal cyst.

posits in, or swellings of the bursae, such as the subdeltoid, radiohumeral, olecranon, trochanteric, prepatellar, and Achilles. Cyst of the lateral meniscus of the knee (Fig. 1) is often mentioned as a possible diagnosis because of a sharply defined soft tissue bulge at the lateral margin of the knee joint. This should be differentiated from general synovitis, in which condition the bulge is bilateral and the suprapatellar pouch is usually distended with exudate.

A thickened infrapatellar fat pad can be

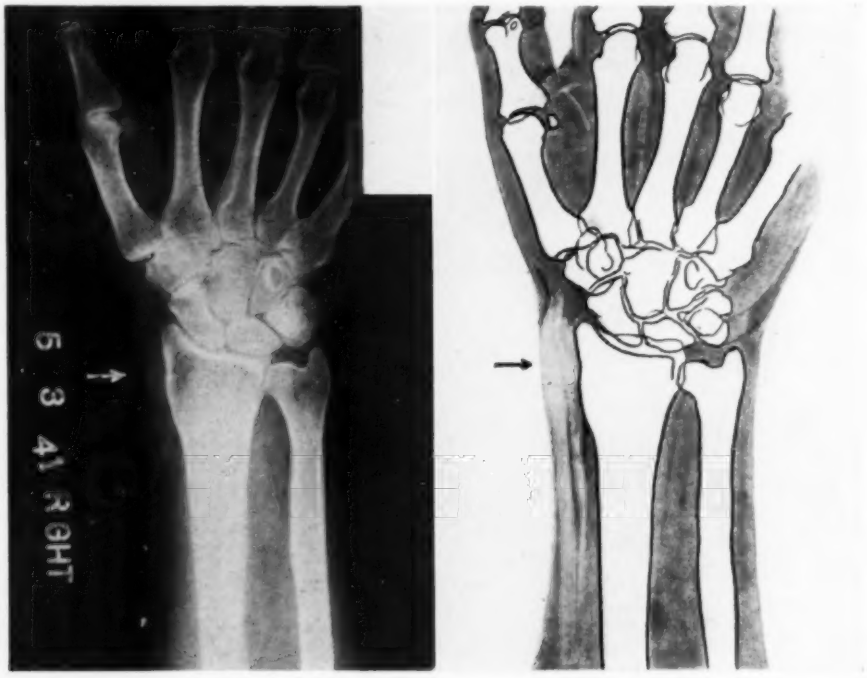

Fig. 4. Tenosynovitis lateral to lower extremity of radius. Characterized by blurred soft tissue swelling, blotting out the shadows of the tendons.

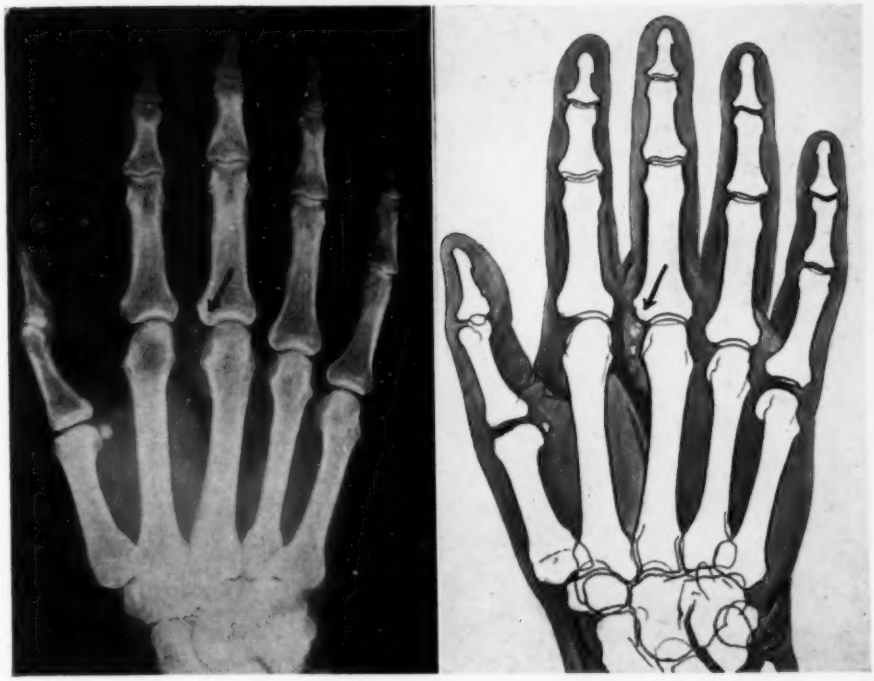

Fig. 5. Inflammatory deposits of lime of varying density, probably along tendon of interosseous muscle. Analogous to the condition found frequently along the supraspinatus tendon.

recognized, and one of these removed in this hospital was found to contain a cyst (Fig. 2). A popliteal cyst is sometimes visible, though the heads of the gastrocnemii with overlapping hamstring tendons often obscure it (Fig. 3). Inflammation about tendons, especially along those lateral to the lower extremity of the radius, is frequently suggested by hazy soft tissue swelling in this region (Fig. 4). Inflammatory calcareous deposits are laid down along some tendons, most frequently the supraspinatus, less frequently elsewhere (Fig. 5). Ganglia appear at the back of the wrist as localized soft tissue bulges with absence of any evidence of joint pathology. They are less readily identified in other parts of the body.

Good visibility of soft tissue neoplasms is often possible, both extracapsular and intracapsular, and their nature can be suspected in some cases from their location, density, and structure. In the knee joint the more common neoplasms are xanthomas,

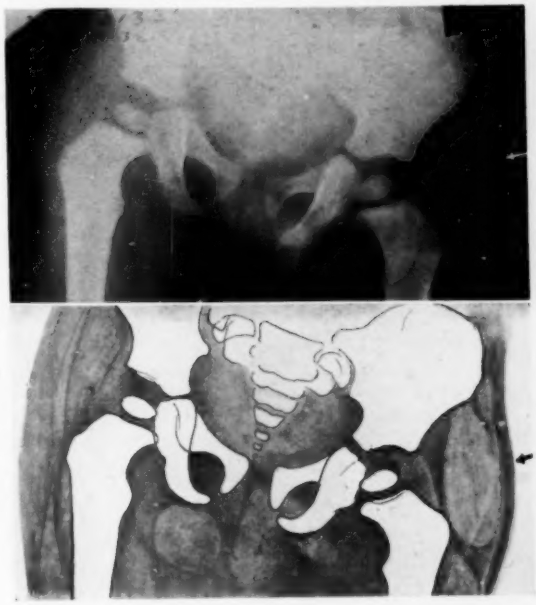

Fig. 6. Hemangioma. Recognized in this examination only as a soft tissue mass. Identified as an angioma after injection with diodrast. Diagnosis established by operation.

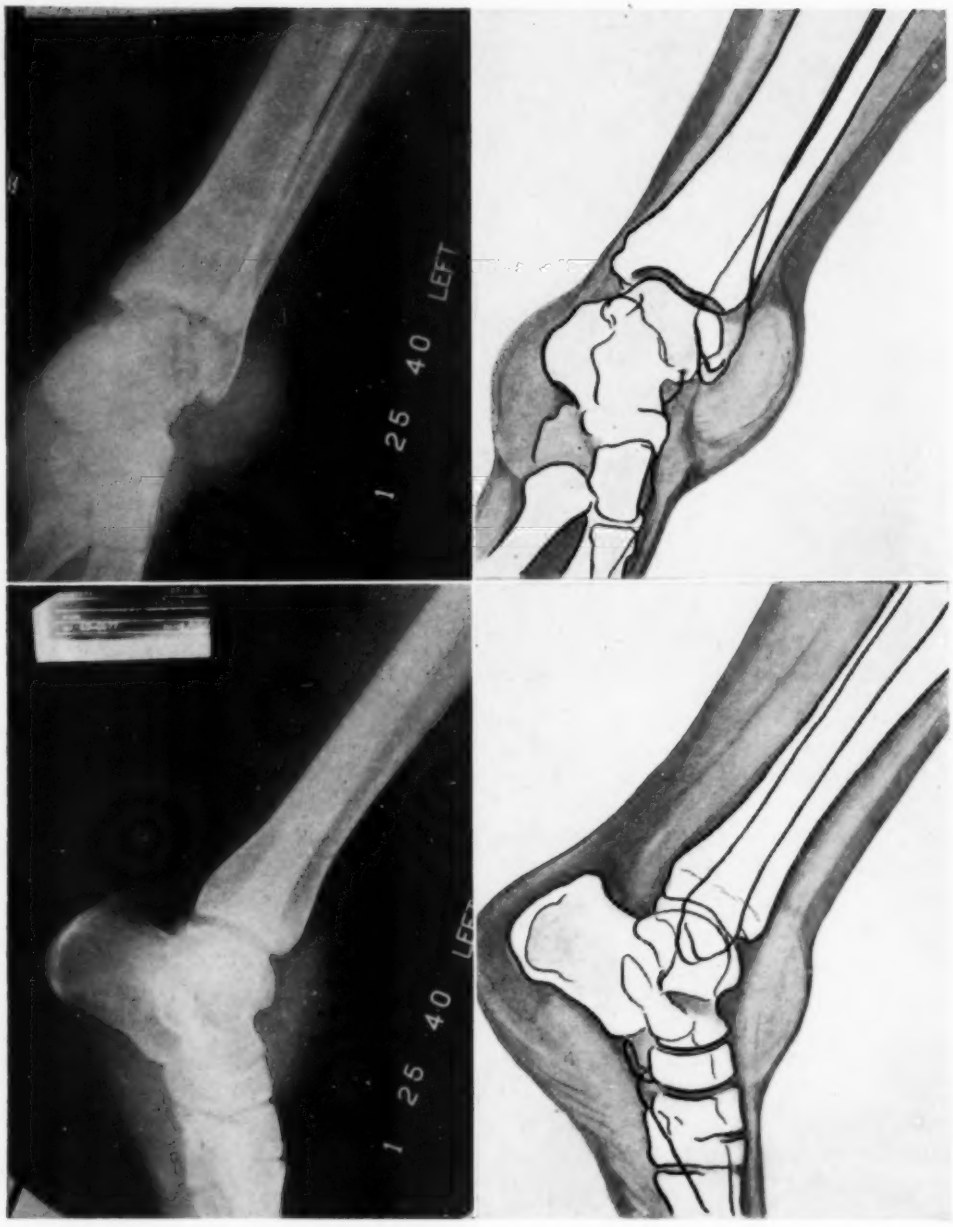

Fig. 7. Lymphangioma. Recognized only as a soft tissue tumor mass.

angiomas, and synoviomas. These can be recognized as tumor masses when there is not too much associated synovitis, but further differential diagnosis is not generally possible (Fig. 6, 7, and 8). When a synovioma is lime-containing, it is easily diagnosed.[7]

In traumatic cases, sprains show subcutaneous swelling in characteristic regions, without capsular distention as a rule, and without evidence of bone injury. Synovitis is manifested by capsular distention, accompanied in the knee joint by a pyriform mass in the suprapatellar pouch. Great assistance can be given the clinician in many cases by differentiating between intracapsular and extracapsular injury, between synovitis and bursitis or contusion (Fig.

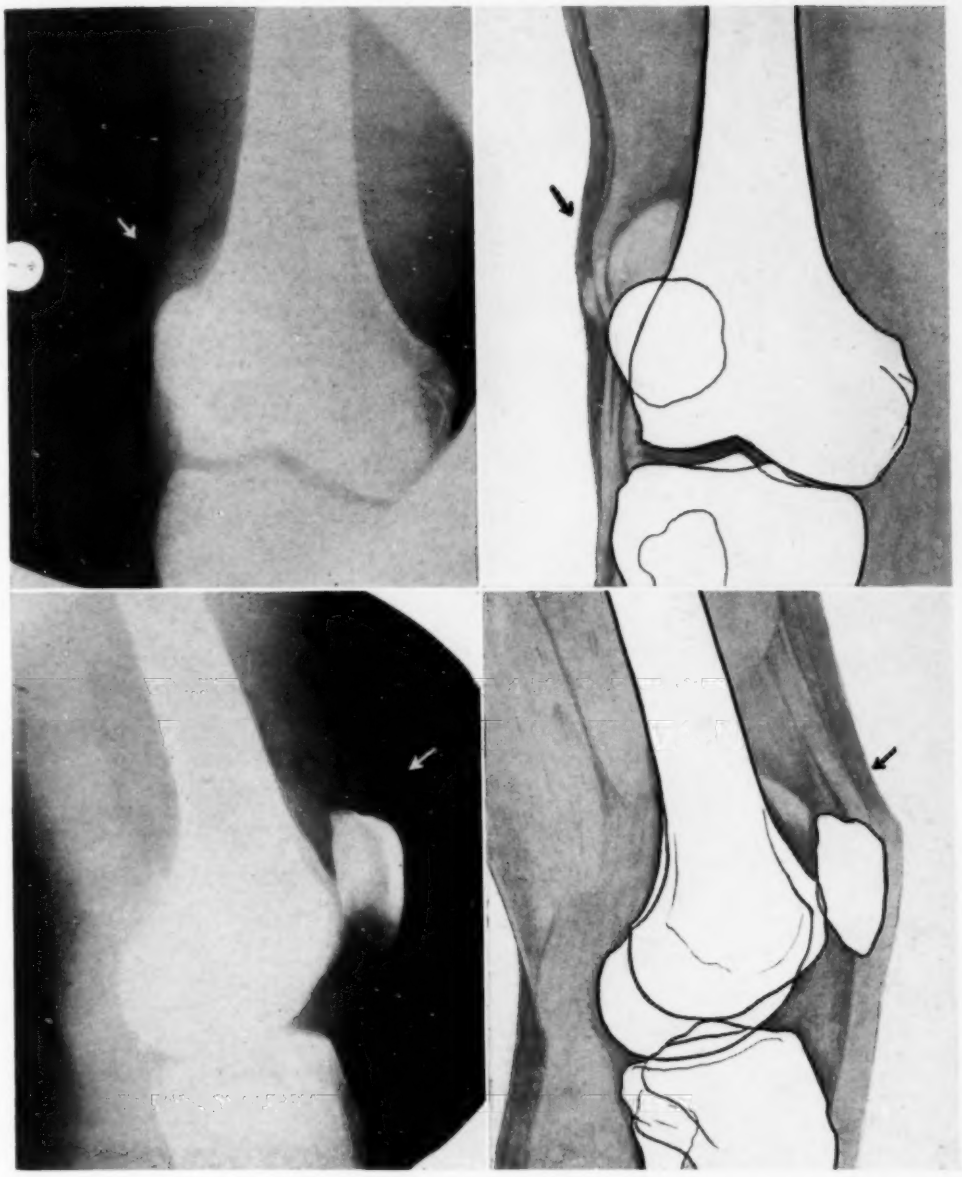

Fig. 8. Hemangioma in knee joint. Preoperative roentgen tentative diagnosis, synovioma or angioma.

9). Tears of the triceps, quadriceps, and Achilles tendons are indicated by absence of the tendon shadow in the region of injury, with marked swelling and clouding produced by hemorrhage and edema. A particle of bone torn off and retracted with the tendon confirms the diagnosis (Fig. 11).

Soft subperiosteal calcification is the most frequent early manifestation of march foot, of the so-called pseudofracture of the tibia described by Roberts and Vogt,[8] and of acute osteomyelitis. Dense films will give no clue to these important conditions. Nor will dense films show foreign bodies of relatively slight density, such as glass and the graphite point of a pencil. This may not be of such great clinical importance in certain portions of the body; but demonstrating

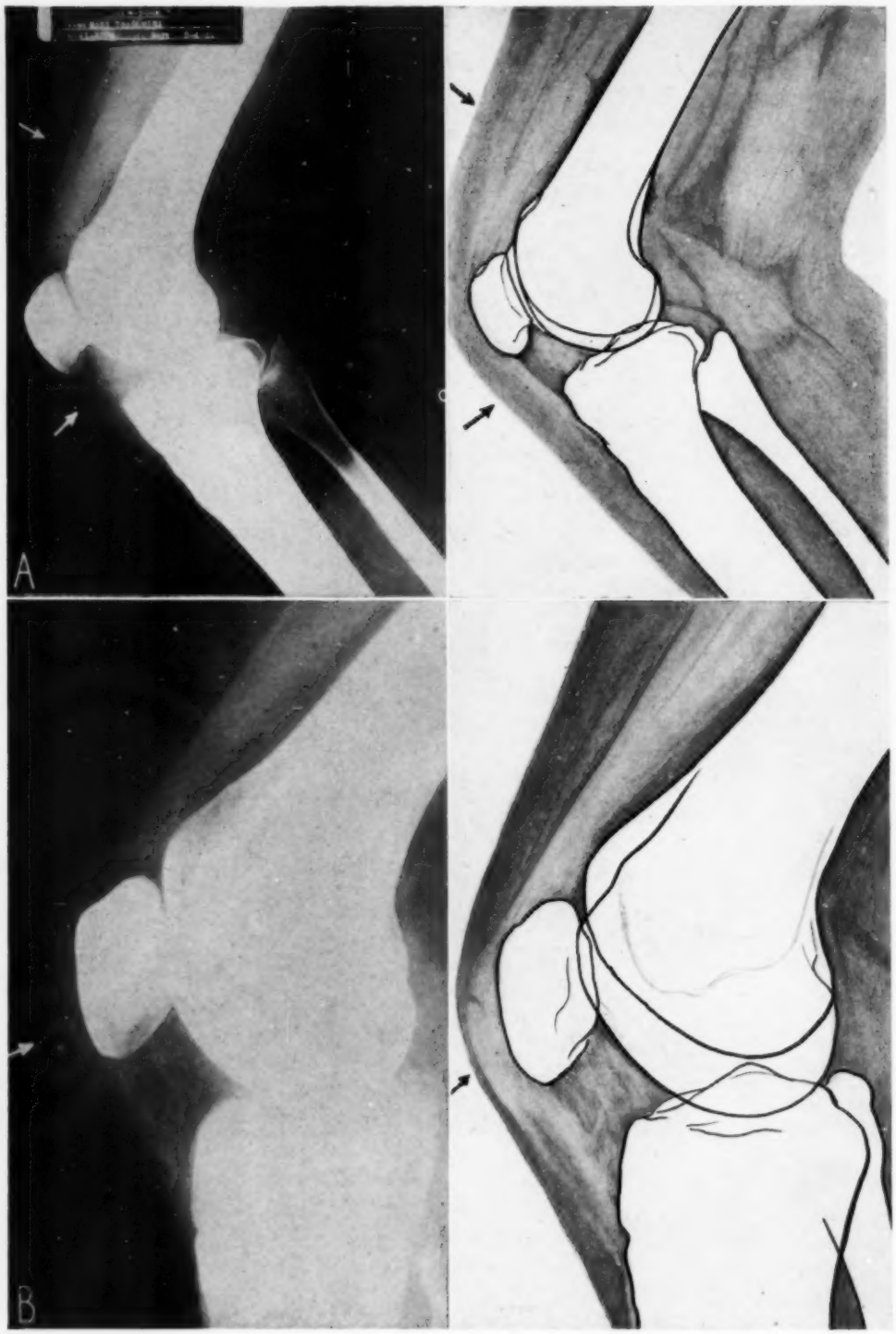

Fig. 9. Usually easy differential diagnosis between intracapsular and extracapsular exudate. *A*, synovitis: pyriform exudate in suprapatellar pouch, thickening of shadows in infrapatellar portion of joint, bulging of patellar ligament. *B*, prepatellar bursitis: suprapatellar and infrapatellar portions of joint clear, no bulging of patellar ligament, localized subcutaneous thickening anterior to patella.

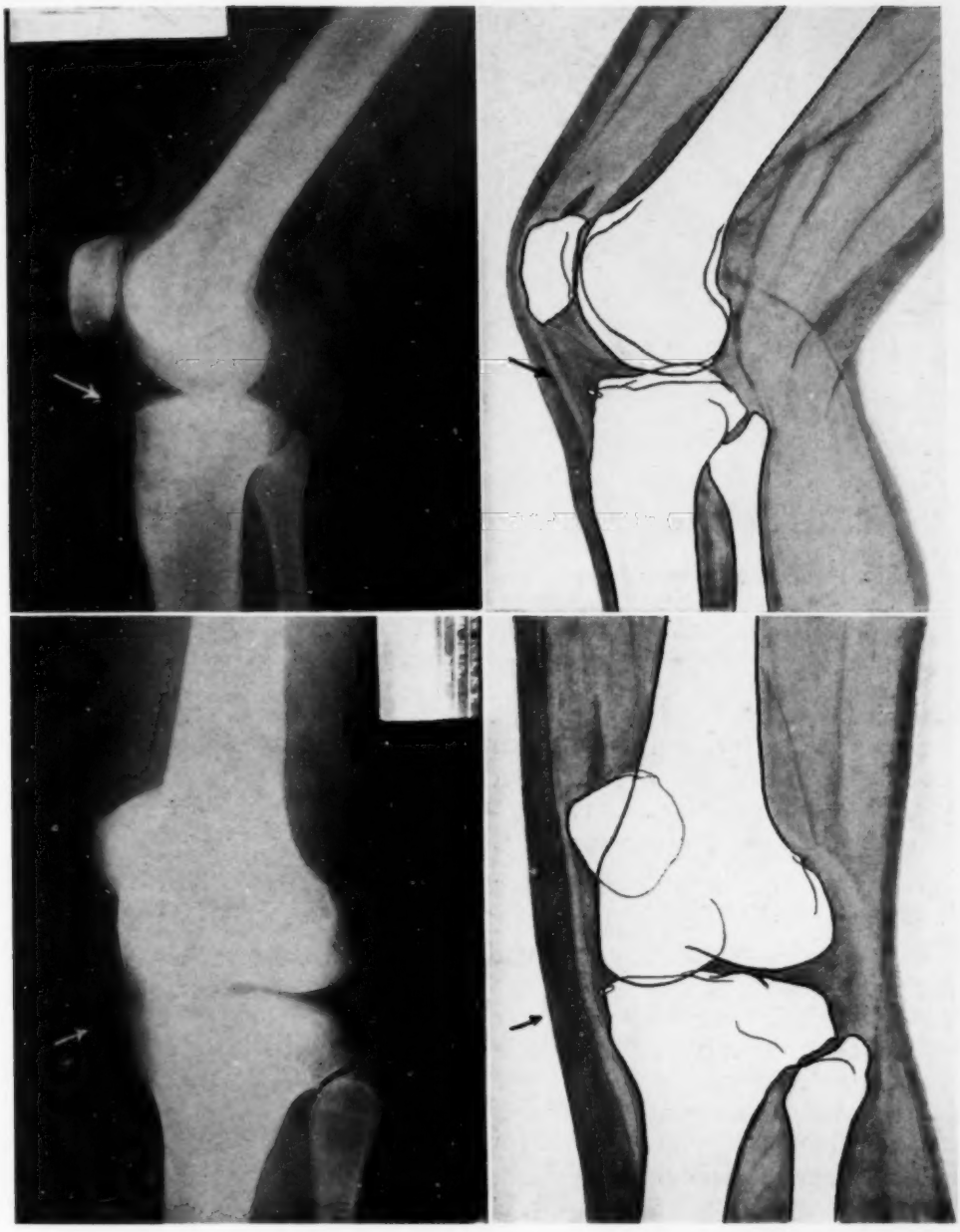

Fig. 10. Graphite point of lead pencil as foreign body in knee joint. Correctly diagnosed before operation, after consideration of the history and making experimental roentgenograms of lead pencils.

that such foreign bodies were the cause of acute synovitis in knee joints was of major significance (Fig. 10).

Subcutaneous varicosities are clearly visible in roentgenograms of proper density; a blurred density and swelling in relation to an enlarged vein is strong evidence of phlebitis. Angiomas are sometimes recognizable as more or less localized collections of tortuous veins. The presence of numerous phleboliths is confirmatory evidence. In cases of hypertrophy of an extremity or

Fig. 11. Rupture of quadriceps tendon. Marked blurred soft tissue swelling above patella with complete absence of the shadow of the quadriceps tendon; irregular piece of bone torn from upper border of patella and retracted upward; relaxed wavy patellar ligament.

of a portion of an extremity, thickened soft tissues with peculiar pattern have given clues to the cause of the hypertrophy, namely arteriovenous aneurysm, hemangioma, lymphangioma, or neurofibromatosis.

SUMMARY

1. Roentgen study of soft tissues is not sufficiently appreciated or practiced.

2. In an orthopedic hospital the majority of roentgen diagnoses are based wholly or in part on soft tissue findings.

3. Bones and soft tissues can be studied satisfactorily on the same film provided (1) the films are not made unnecessarily dark, (2) illumination of sufficient intensity is used in studying the films. A simple lighting fixture for studying soft tissues in films of greater density is described.

4. Frequently encountered conditions in which soft tissue study has aided diagnosis in an orthopedic hospital are listed, discussed, and illustrated.

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NORTH AMERICAN CUTANEOUS BLASTOMYCOSIS TREATED WITH SUPERFICIAL ROENTGEN THERAPY

A REPORT OF FOUR CASES*

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NORTH AMERICAN blastomycosis (Gilchrist's disease)[8,9,10] is a chronic inflammatory infectious disease produced by *Blastomyces dermatitidis*,[8,12,25] a mold-like fungus. This entity is to be distinguished from other types of blastomycosis, especially the European type caused by the yeast-like fungus, *Cryptococcus hominis*.

Gilchrist's disease has been mentioned in the literature many times and was extensively reviewed by Martin and Smith,[26] and Hyde and Ricketts[18] in their reports. It has been separated into two distinct types:[3] (1) systemic and (2) cutaneous, or localized. Very few detailed reports of roentgen therapy in the cutaneous type have been given in the literature, although many reports mention that superficial roentgen therapy is a method of treatment.[6,17,26,34] Our purpose in this report is to summarize briefly 4 cases treated with superficial roentgen therapy and suggest what we found to be a practical method of handling this extremely resistant and chronic condition.

The usual source of infection in man is not clearly established. Reported cases have been predominantly in the eastern half of the United States.[26]

The onset of cutaneous blastomycosis is characterized by a primary lesion consisting of a papule or papulopustule occurring usually on exposed or injured surface areas.[13,18,26,33] The face is most commonly involved;[35] next most common sites are the hands, arms, and lower extremities, in the order named.[26]

The primary lesion is easily mistaken for a furuncle. But instead of healing it spreads slowly peripherally with direct extension, undermining the epidermis and producing a well defined border surrounding a verrucous or crusted surface plaque[3] (Fig. 1). Spread occurs slowly over a period of months and in many respects simulates epithelioma development (Fig. 6). The plaque is recognizable as a crusted, irregular, wart-like, fissured surface which bleeds easily, is violaceous or purplish-red, and covered with tiny yellow abscesses. These abscesses yield a mucopurulent material usually containing numerous fungi.[25] There is definite tendency towards healing centrally as the lesion ages with formation of a light-colored atrophic scar. The periphery continues a gradual relentless extension (Fig. 8 and 9), which may result in large and grotesque lesions (Fig. 1).

Gummatous type lesions may occur wherein the involvement is in the more internal layers of the corium and subcutaneous tissue.[3] Two types of gummatous development are distinguishable: (*a*) the initially localized primary cutaneous papulopustule which later spreads deeper by direct extension, and (*b*) the breakdown and ulceration of subcutaneous lesions produced by hematogenous spread in systemic blastomycosis.

The gummatous type of lesion appears as a soft, deep-red, nodular reaction surrounded by a reddish-purple unelevated area of skin. The tendency is for multiple similar nearby nodules to develop with eventual breakdown, ulceration, and subsequent formation of the typical border reaction already described (Fig. 1, 5, 8 and 12).

Although systemic disease may occur, usually the cutaneous or localized form of blastomycosis runs a chronic benign course

* From the Departments of Radiology and Dermatology of Duke University Hospital under the direction of Robert J. Reeves, M.D., and J. Lamar Callaway, M.D.

without regional lymph node involvement or systemic reaction.[11,12] Most systemic cases are likely to produce dermal implantations.*[,13] Secondary infection with pus-forming cocci and surface organisms may occur and is partially responsible for the local pain and tenderness and foul odor. Pure blastomycetic lesions are practically without odor or pain.[3,12,26]

Diagnosis is not difficult and is ascertained through (1) history and appearance of the lesion, (2) potassium hydroxide slide preparation[25] of the organism from the tissus borders, (3) biopsy, (4) culture,[25] (5) intradermal test,[25] and (6) complement-fixation reaction.[24]

Potassium hydroxide smear is accomplished by transferring mucopurulent material from the lesion border to a clean slide, adding several drops of 10 per cent potassium hydroxide solution, covering with a cover slip and allowing to stand several minutes before examining with a microscope for the characteristic budding organism.[25]

Biopsy, which has no contraindication, reveals irregular papilliform projections with hyperkeratotic plugs.[2,35] The epidermal rete forms inward projections and is definitely acanthotic. Characteristic miliary abscesses may be seen in the epithelial prolongations and in the corium.[2,12] The epidermis contains numerous collections of agranulocytic leukocytes, with the dermis showing considerable infiltration of polymorphonuclear, epithelioid, and plasma cells with edema. Giant cells with or without *Blastomyces* may be found also in the dermis. Staining with polychrome methylene blue or hematoxylin and eosin is satisfactory.[25,33] Budding forms of the organism may be seen in the tissue but never the hyphae.[11,33] Subcutaneous lesions reveal a periphery of plasma cells, mast cells, lymphocytes, and fibroblasts surrounding a zone of Langhans type giant cells which in turn surround a center of necrotic débris, leukocytes and organisms. This reaction is not unlike that of tuberculosis.[2]

* Two such cases have recently come under our observation.

The fungus develops on Sabouraud's medium at room temperature as a white cottony, mold-like, aerial growth. Microscopically, chlamydospores and many branched hyphae bearing lateral conidia are characteristic.[25] If planted on blood agar at 37° C. it grows slowly as a small, waxy wrinkled or porcelain-like colony. Microscopically a smear of this material shows round or oval, greatly refractile bodies, about 10 to 20 microns in size[33] with a double rimmed capsule. Budding forms and small hypae may be seen. The mold-like growth on Sabouraud's medium at 20° C., when transplanted to blood agar at 37° C., grows as a waxy, crinkled colony and then may be retransplanted to form mold-like colonies on Sabouraud's medium at 20° C.[26] Two weeks or longer is usually required for growth,[25] and cultures should not be discarded as negative unless this time has elapsed.

The intradermal test is accomplished by injecting into the skin 0.1 cc. of the heat-killed vaccine and interpreting the reaction at twelve, twenty-four, and forty-eight hours, just as in an ordinary tuberculin test. The complement-fixation reaction is usually negative in the purely localized type of cutaneous blastomycosis unless the lesion is very extensive.[24]

Differential diagnosis involves distinguishing the condition from cancer, epithelioma, late syphilids, sporotrichosis, tuberculosis of the skin including tuberculosis verrucosa cutis, granuloma inguinale, bromoderma, ioderma, and other granulomas.

Prognosis for recovery is good with recurrences not uncommon. Early diagnosis and treatment minimizes the inevitable scarring and disfigurement (Fig. 4).

MEDICAL TREATMENT

Treatments mentioned in the literature include excision, electrocoagulation, surgical diathermy, and many others. Administration of iodides and superficial roentgen therapy are definitely efficacious, particularly in combination, and the two represent the treatment of choice, we believe, pro-

vided the patient is not allergic to the fungus since potassium iodide is extremely dangerous to the hypersensitive patient.[3,26,31] General hygienic measures should not be neglected.

The importance of determining the state of hypersensitivity by the intradermal test cannot be overemphasized.[3,26] Reduction of the allergic reaction is accomplished by administration of small doses of heat-killed vaccine. The doses are increased progressively until a skin test indicates a decrease in the allergic state. It is impossible to eliminate the allergy entirely, but unless this desensitization is carried out before the administration of iodide, dissemination of the disease may occur.[3,26] The spread is thought to be comparable to the dissemination of tuberculosis by administration of iodide. After some reduction of the hypersensitivity has been accomplished, iodides can be started cautiously through the oral route, using gradually increasing doses of an aqueous solution of potassium iodide. Supplementary intravenous iodide may be administered with 10 to 100 cc. of a 10 per cent solution of sodium iodide injected daily, but the intravenous route is rarely if ever necessary.

ROENTGEN THERAPY

Superficial roentgen therapy, in conjunction with the medical measures already outlined, stops activity within the spreading borders, eliminates secondary infection, and hastens regression of the chronic inflammatory reaction. The sooner roentgen therapy is started, the less deforming will be the scar. Even in the patient sensitive to his own organism there is no logical contraindication to roentgen therapy while being desensitized. It embraces all the principles of superficial roentgen therapy for inflammations,[1,5,6,7,14–17,19–23,28,32,34] and probably acts as a stimulatory agent to the local tissue resistance promoting repair through scarring. After as little as 300 r is given the entire inflammatory process changes and the pathological picture becomes one of definite healing through fibrosis and regeneration of epithelium (Fig. 8, 9, 15 and 16).

Small doses of superficial roentgen rays, 100 kv., constant potential, 8 ma. (inherent oil filtration equivalent to 3 mm. Al*), half-value layer 3.20 mm. Al, measured in air, without backscatter, and half-value layer 3.40 mm. Al, with backscatter, were used in our cases.† Fifteen centimeter distance was used instead of greater target-skin distance to decrease the percentage depth dose. The piled up, elevated, thickened, verrucous character of the borders makes this quality ray desirable to assure penetration into the depths of the dermis. Thus, the quality radiation we used was not the purely soft unfiltered ray of longer wave length from an air-cooled glass bowl tube. Doses hereinafter referred to are in terms of roentgens in air applied to the skin without backscatter.

Ideal roentgen therapy should be a series of single treatments of 75–100 r per treatment, administered at weekly or ten day intervals, for a total dosage sufficient to produce smoothing of the borders and remove signs of activity (Fig. 2, 4, 11 and 16). The total dosage required varies with each individual and the seven to ten day interval between treatments is advisable in order to prevent cumulative effect and overdosage; 1,500 r or less should be adequate, if our cases are illustrations of proper management.

Progress in healing and repair is easily ascertained with an interval schedule such as we have mentioned. The first obvious effect of irradiation is drying of the central portion. Then the foul odor disappears, the erythematous granulation blanches, and the peripheral border subsequently begins to become smooth (Fig. 11). When there is evidence of healing at the center, this dry and flat scar is carefully shielded with lead foil on subsequent treatments so that only the borders receive additional irradiation (Fig. 16). Repigmentation in islands of the

* Westinghouse factory calibration data for tube.

† Checked by J. E. Morgan, Ph.D., registered roentgen-ray physicist.

central portion begins early (Fig. 2 and 4), and progresses steadily during and after treatment (Fig. 4, 7, 11 and 16). As the border becomes level, repigmentation progresses on the side toward the healing center. A margin of ½ to 1 inch of normal skin beyond the active border is sufficient, and this margin can be reduced as treatment progresses. In our cases the central portion of the lesion required very little of the total roentgens given and certain portions of the borders required more than other portions (Cases I, II and III). This unequal distribution of dosage required for different sections of the lesion can be individualized for each patient at each treatment and is not difficult to evaluate.

As soon as there is tendency to drying of the central scar and when odor is almost gone, we suggest daily application of olive oil or cottonseed oil locally to the center. This combats the dryness produced by the rapidly healing scar tissue and adds considerably to the comfort of the patient.

Cautious treatment with extended intervals of two or more weeks, if necessary, between each application may be desirable in some grotesque cases because of the extreme radiosensitivity of the lesions. Such an interval schedule would have been preferable in Case I. This patient received probably twice as much (or even more) total roentgens than was necessary to his eyelids. Consecutive daily treatments to his eyelids were administered in his second series allowing an unpredictable cumulative effect. A method of therapy of that type is poor treatment and certainly leads to overdosage with inevitable undesirable sequelae, i.e. overproduction of scar tissue, telangiectasia, etc. Undertreatment will just as surely result in early false response with later recurrence (Case IV). With, however, the proper interval between single applications of superficial roentgen therapy, gradual response can be evaluated and the immunological status can be followed. Undertreatment should thereby be prevented just as precisely as overtreatment can be avoided.

Most cases of Gilchrist's disease will respond to therapy such as we have outlined. However, too much optimism about the value of iodides and radiation therapy, especially in more deeply seated lesions where there is involvement of bone, is not justified without intensive immunological studies. For example, one such patient with cutaneous blastomycosis extending into the ulna did not improve with roentgen therapy and iodide until immune rabbit serum was administered.[27] Thus an adequate study of the immunological picture is equally deserving of attention and cannot be disregarded as a factor in the general plan for therapy.

CASE REPORTS

CASE I. A. J., male, colored, aged twenty-five, presented himself in the Medical Out Patient Clinic on January 3, 1941, with a chief complaint of "spreading sore" left side of his face of one year and nine months' duration.

The initial lesion started as a hard thickened area about 1 inch in diameter on the middle of the left cheek with a white center which he squeezed, expressing a small amount of pus. The area then became ulcerated at its center, was tender, and his family doctor cauterized it. New pustules appeared about the periphery of the cauterized area and again his local physician cauterized each of these. Two weeks later, the lesion was four times its original size, his whole left face was swollen and tender, and there was a "kernel" in the neck at the angle of the jaw. The physician incised this node and inserted a drain, but there was no drainage so the drain was removed. Within six months, in spite of various antiseptics applied daily the ulceration had extended to the left temporal region. During this period another lesion developed at the angle of the left jaw where the drain had been placed and this ulceration spread rapidly with a wart-like border. There was such foul odor that members of the house could not tolerate the patient's presence in the same room. Three months before admission, the two lesions joined and spread to the left eyelids with much edema and closure (Fig. 1).

Laboratory findings were negative except for positive cultures for *Blastomyces dermatitidis* and secondary pus organisms. Biopsy on January 10, 1941, was reported "blastomycosis."

The patient was hospitalized. Since he gave

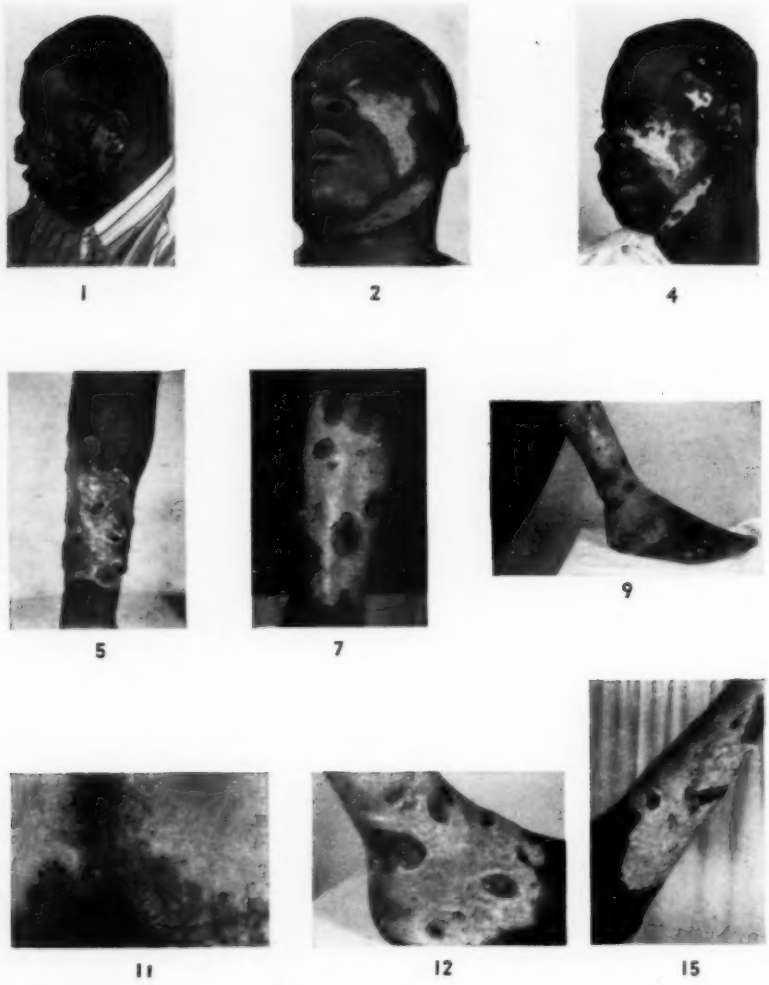

FIG. 1. Case I. Active blastomycosis of twenty-one months' duration showing typical pseudo-epitheliomatous border before therapy. Patient sensitive to organism.

FIG. 2. Case I. Six weeks after first series of roentgen therapy. Borders have smoothed, repigmentation beginning. Eyelids edematous and active infection present in external ear.

FIG. 4. Case I. Fourteen weeks after first series and eight weeks after the additional irradiation to eyelid and ear of the second series. Repigmentation is striking but eyelids are scarred from cumulative overdosage.

FIG. 5. Case II. Appearance of leg three years after initial lesion and two months before roentgen irradiation started.

FIG. 7. Case II. Four months after roentgen therapy showing dry, scaly, healed scar with repigmentation islands and level borders.

FIG. 9. Case III. Active blastomycosis seven years old before roentgen treatment started. The attempt at healing centrally has interfered with blood supply to foot. Active granulations evident at borders.

FIG. 11. Case III. Close-up of treated border of Figure 10 after 750 r, showing rapid response of active granulation with beginning repigmentation.

FIG. 12. Case III. Ankle after 1,000 r to granulations of Figure 9. The two granulations on dorsum of foot received only 750 r and have not sloughed.

FIG. 15. Case III. Area shown in Figures 10, 11 and 14 four weeks after Figure 14. During this month the patient received no additional irradiation and the sloughed areas of Figure 14 are healed.

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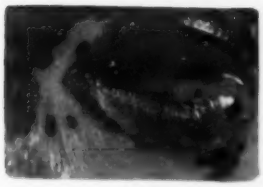

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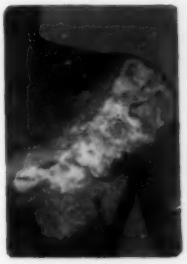

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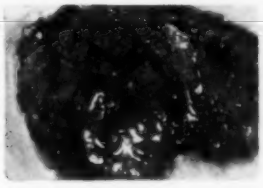

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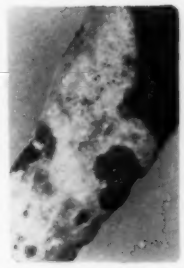

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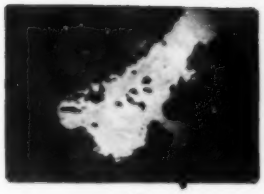

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Fig. 3. Case I. Close up of edematous eyelids six weeks after first series of roentgen therapy; an additional 1,500 r given to this area was probably overdosage and not needed.

Fig. 6. Case II. Two months after medical treatment (densensitization and potassium iodide). Definite activity at borders with pseudo-epitheliomatous granulations but some healing centrally under medical therapy alone.

Fig. 8. Case III. Active blastomycosis of shoulder and axilla seven years old when roentgen treatment was started.

Fig. 10. Case III. Typical active granulation at border with central scarring seven years after initial infection.

Fig. 13. Case III. Close-up of ulcer where largest granulation sloughed after 1,000 r.

Fig. 14. Case III. Area of Figures 10 and 11 after 1,000 r. A portion of the peripheral granulation sloughed, actual border well healed and smooth.

Fig. 16. Case III. Healed shoulder and axilla eight weeks after roentgen therapy. The center of this lesion received only 300 r, the borders 600 r. Compare with Figure 8.

Fig. 17. Case IV. Active blastomycosis of the thigh four years old before therapy. The biopsy suture can be seen at the border.

Fig. 18. Case IV. At completion of series of roentgen therapy, 750 r. Borders smoothed but center not entirely scarred or healed. This lesion was entirely healed six weeks after this picture was taken.

a 3+ positive skin test he was desensitized with *Blastomyces* vaccine beginning on January 7, 1941, and continuing through March 1, 1941. Potassium iodide orally was started January 21, 1941, when the immunological picture by intradermal test warranted it, and continued during the whole series of roentgen treatment. Superficial roentgen therapy was started January 22, 1941, 100 kv. constant potential, 8 ma., 15 cm. distance with inherent oil filtration equivalent to 3 mm. Al.

Using 150 r dosage every four to seven days, radiation was administered for a total of 2,100 r, shielding the center after it received 450 r. Some portions of the borders received only 950 r, but the eyelids and bridge of the nose received the full 2,100 r. Response was excellent and the patient was discharged on March 3, 1941.

He returned April 22, 1941 (Fig. 2), with activity in the eyelids and external auditory canal. These were given additional therapy with 100 kv. constant potential, 8 ma., 4 mm. Al filter in addition to the inherent oil filtration equivalent to 3 mm. Al, at a 30 cm. distance with small cone. The eyelids received 1,500 r and the ear 1,200 r over a period of eight consecutive days. This patient was last seen June 23, 1942 (Fig. 4), with no evidence of recurrence, but a fibrosis of the eyelids causing partial closure. This scarring which includes the left tear duct is partially the result of overdosage to the eyelids and makes desirable a plastic repair at some future date if the patient is to have use of the eye which when last seen had 20/20 vision.

CASE II. J. H., male, colored, aged twenty-eight, was first seen in the Surgical Out Patient Clinic November 8, 1938, complaining of "sores of the left leg" which had been present for three years and were unhealed after five treatments by a local physician who applied "caustic."

The initial lesion was a furuncle with a whitish center. He squeezed this several times and pus was expressed, but the sore spread continuously, ulcerated, and would not heal. The physician's "caustic" applications produced a thick scab each time but the skin never grew under the scab and when the scab was washed off the ulcer remained. The sores had continued to enlarge, ulcerate, and joined other sores, with an offensive odor in spite of all attempted remedies.

Laboratory studies were negative except that a smear directly from the lesion border showed double contoured and budding yeast-like forms of *Blastomyces* in considerable quantity.

The patient was hospitalized and skin test was negative. He was placed on a house diet supplemented with cod liver oil, tomato juice, brewer's yeast. The ulcers on the leg were compressed with normal saline. He received potassium iodide orally, 3 drops t. i. d., increasing 1 drop per dose so that he was receiving a total of 39 drops of an aqueous solution at discharge November 22, 1938. The ulcers cleared of secondary infection and appeared to be healing at the center.

On January 17, 1939, the patient returned with definite evidence of activity in spite of regular surgical diathermy treatments (Fig. 6). Superficial roentgen therapy was started January 18, 1939, because the medical measures and diathermy had failed to heal the lesions and stop the spread peripherally. Four single treatments (100 kv. constant potential, 8 ma. 15 cm. distance, 4 mm. Al filter) of 200 r each treatment were given over a period of two months with an interval of two weeks between treatments. The lesions healed centrally and the borders regressed. The series was stopped March, 1939. On December 2, 1940, the patient returned with recurrence in the old lesion borders about the ankle. A series of six more treatments, each of 200 r shielding the center, was administered with two to four week intervals between treatments completing a second series total of 1,200 r on March 17, 1941. This patient was followed closely from April through July, 1941, and showed no evidence of recurrence July 19, 1941, when last seen (Fig. 7).

CASE III. N. A., male, colored, aged twenty-five, presented himself in the Out Patient Clinic December 29, 1936, with a chief complaint of "sore" on the right foot and left shoulder of two and one-half years' duration. The initial lesions were small furuncles which developed simultaneously over the anterior margin of the left shoulder and over the lateral portion of the right ankle. He squeezed these and very little pus was expressed. The two "sores" would not heal, ulcerated and spread rapidly. He saw no physician and had used only various "antiseptics." There was very little pain, but the ulceration about the ankle became so large that he experienced difficulty in walking and had to discard his right shoe.

Biopsy of the skin lesion revealed "blas-

tomycosis"; culture from the borders of the acilla and ankle were positive for *Blastomyces dermatitidis*, and the characteristic organisms were also found on direct potassium hydroxide smears. Intradermal test for sensitivity to *Blastomyces* was strongly positive.

Copper sulfate compresses and desensitization with *Blastomyces* polysaccharides[31] for twenty days did not alter the lesions. After desensitization potassium iodide orally and iontophoresis (copper sulfate) were continued for ten days and the patient was discharged from the hospital showing little improvement. He received eight more treatments with copper sulfate iontophoresis during the following six weeks and continued to take potassium iodide orally but showed no improvement.

Four years later he returned and stated that the areas had spread continually in spite of "all kinds of medicines" which, however, did not include iodides, administered at one of the State penitentiaries. The appearance at this time was as shown in Figures 8, 9 and 10.

Superficial roentgen therapy was started (100 kv. constant potential, 8 ma., 15 cm. distance with an inherent oil filtration equivalent to 3 mm. Al) and after 300 r in divided doses the axilla began to heal. The center was then shielded and the borders of the axilla and shoulder received an additional 300 r before they smoothed down (Fig. 12, 13 and 14). This patient is still under observation and when last seen in August, 1942, the craters on the leg where the granulations had sloughed were entirely healed (Fig. 15), and the shoulder and axilla were also healed (Fig. 16).

Case IV. N. G., male, white, aged fifty-one, entered the Surgical Out Patient Clinic May 5, 1941, with chief complaint of "skin cancer," right thigh, of thirteen years' duration (Fig. 17 and 18).

The first lesion was a sore composed of several small pustules over the buttocks. These soon ulcerated and the borders spread. Four months after onset a painless, hard, immovable nodule appeared in the right gluteal fold. This remained unchanged and asymptomatic for about two years and then became eroded and was covered with a thick crust. The ulcerated area spread until it covered the entire gluteal fold or an area about the size of his hand. He received five small roentgen treatments of unknown quantity in 1932 with some immediate improvement but never complete healing.

Intradermal test for sensitivity to *Blastomyces* was strongly positive. Biopsy was also positive for blastomycosis.

He was hospitalized and desensitized to *Blastomyces* antigen. Potassium iodide was then started orally. On May 16, 1941, superficial roentgen therapy was started and over a period of four weeks 975 r in divided doses was administered, with a rapid regression of the lesion. When seen six months after therapy was completed there was no sign of activity and the previously ulcerated area was covered with a clean smooth scar.

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Vol. 48, No. 5

TOTAL BODY IRRADIATION

WITH REVIEW OF CASES

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FOR many years, the oncologist has sought a method of therapy whereby cancer widely disseminated through the body might be destroyed, or whereby, with the local eradication of cancer by surgery and irradiation, future metastatic recurrence of the disease might be prevented. It is a well known truism that localized cancer can usually be completely eradicated but subsequent distant metastases cause death by destruction of vital organs. Therefore, it was only logical that irradiation of the entire body should have been first employed with the hope of devitalizing or destroying all wandering malignant cells or early metastatic foci without destruction of the host. The basic principles of the method of total body irradiation were first described in the form of an "x-ray bath" by Dessauer[11] in 1907 but were not applied until 1923 when Chaoul and Lange[3] reported favorable results in 12 cases of Hodgkin's disease treated with small doses of roentgen rays to the entire body. Although Teschendorf's[33] results of treatment of leukemia and Hodgkin's disease by total body irradiation were first reported in 1927, he is given credit for having introduced this method of therapy in Europe where it is known as the "Teschendorf method." Similarly, at Memorial Hospital in 1931, Dr. Arthur C. Heublein[13] initiated treatment by continuous irradiation of the whole body for a long duration and the method has come to be known as "Heublein therapy."

Following the first reports of favorable responses in cases of Hodgkin's disease and leukemia, experimental animals were similarly treated in order to evaluate the biologic effects and rationale of this therapy. Kok and Vorlaender[16] and Caspari[2] induced regression of tumor masses in animals by smaller doses of generalized irradiation than was possible by local treatment. Murphy and Nakahara[19] in 1922, reported the production of an immunity against cancer in mice given general exposure to roentgen rays and also inoculated with tumor cells. In a study of the lethal effect, Nater and Schinz[20] reported on the basis of animal studies that the application of one erythema dose to the whole body produced rapid death. These experimental observations were soon supported by further clinical studies (Sluys [30,31] and Mallet[17]) which showed, in general, deleterious effects, often resulting in increased tumor growth or rapid death of the patient when large doses of roentgen rays were applied to the body. On the other hand, with small doses, the health of the patient was often greatly improved and at the same time the tumor growth was restrained. The latter observation constitutes the primary rationale of this form of therapy. In a discussion of the constitutional reaction to roentgen therapy, Ewing[12] in 1926 stated that the nature of the favorable reaction on the part of the body as a whole was complex and little understood, but probably of much importance in the cure of malignat tumors by irradiation.

Recently, Abels *et al.*[1] have demonstrated a frequent biochemical change in blood cells following external whole body irradiation as developed by our Heublein unit. Following irradiation of a patient, a fluctuation in both red and white cell organic acid-soluble phosphorus was noted for a period of about twelve days. The exact significance of this change in phosphorus metabolism is not known.

The practical application of total body

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irradiation has varied in accordance with the therapeutic equipment and concepts of the various clinics. Teschendorf's[33] method consisted of exposure of the entire body to a hard radiation filtered by 0.5 mm. zinc and 2 mm. aluminum at a distance of 180 cm. for a total daily dose of 3 to 5 per cent of an erythema. Sluys,[30,31] in his reviews of teleroentgen therapy of Hodgkin's disease in 1930 and 1933, reported that the method of total body irradiation at a single treatment had been largely abandoned because of unfavorable reactions, and described a method whereby one of four body quadrants was treated daily with 25 to 50 r produced by a 200 kv. unit at 1.5 meters target-skin distance for a total of 2,000 to 2,400 r to the chest or abdomen. Scott[27] further modified the factors to obtain wide-field roentgen therapy by using a 100 kv. unit at 20 inches target-skin distance with a maximum area of body spread of 22 inches and the optimum dosage 60 to 100 r applied once or twice for six or eight treatments. Dale[9] modified the high voltage therapy technique by increasing the daily dose to as much as 4/5 S.E.D. and shortening the target-skin distance to 1 meter; treatments were carried out for from several days to five weeks, according to the response of the patient. The advantages claimed by the proponents of teleroentgen therapy are that it produces fewer unfavorable constitutional reactions and less tendency to develop anemia.

The increasing number of favorable reports in the European journals of either total body irradiation or prolonged irradiation of the body using large portals (teleroentgen therapy) for many neoplastic diseases is testimony to the usefulness and safeness of this method. It is sufficient to refer to a few of the more important reports and to specify the diseases treated—Hodgkin's disease (Chaoul and Lange,[3] Teschendorf,[33] Sluys,[30,31] Mallet,[17] Scott[27]), leukemias and polycythemia (Salzman,[25] Sgalitzer,[28] Scott[27]), advanced uterine and breast cancer (Mallet[17]). Since 1935 several complete articles on the indications for general body irradiation have been published by Delherm and Stuhl,[10] Cottenot and Sluys,[4] and Scott.[27] There are few previous reports of this method of therapy in American clinics: Heublein's[13] introductory report of the method of therapy with a review of 9 cases; Craver and MacComb[8] reviewed 134 cases treated during the first two year period at the Memorial Hospital, and recently Sanderson[26] reported a group of 22 cases of lymphomatoid diseases treated at the Massachusetts General Hospital. In 1935, O'Brien[22] commented on the use of teleroentgen therapy at the Boston City Hospital, and suggested that the method offered hope of greater salvation in the "lymphomas." Analysis of the literature shows a general agreement that total irradiation produces its best results in the lymphomatoid diseases with few groups reporting favorable palliation of advanced metastatic cancer. Many of the reports, however, concern small groups of cases and the impressions derived therefore are of questionable value.

The present report presents a critical analysis of 270 cases treated at the Memorial Hospital over a ten year period, from 1931 to 1940. The purpose of this study was to obtain, if possible, (1) more exact indications for total body irradiation; (2) the end-results, not only as measured by palliation of the patient, but, in the large groups of Hodgkin's disease, lymphosarcoma, and the leukemias, to determine the survival rates following therapy; (3) to compare survival rates of patients thus treated with those of patients receiving no treatment or given other forms of therapy; (4) to determine the exact dangers and contraindications to this method, and (5) to analyze the various changes in the blood picture following total body irradiation.

Heublein, in collaboration with Craver and Failla of the Memorial Hospital, reasoned that, since anaplastic cells and those in active mitosis are more radiosensitive, total body therapy should involve prolonged, continuous irradiation with hard roentgen rays of low intensity at long target-skin distance. The original roentgen unit, designed by Failla and described by

Craver and MacComb,[7] operated in a four bed ward with the factors 185 kv., 3 ma., 2 mm. copper filter and 5.4 and 7.3 meter distances from the target of the tube to the beds. According to modifications in the new Memorial Hospital, the unit has been placed in the wall between two single rooms and treatments can be carried out in one or both rooms simultaneously, as seen in Figure 1. The following factors are employed: 185 kv., 1 ma., 5.5 mm. copper equivalent, and the distance from the roentgen tube to the patient is 3 meters. The output has been varied from 1.5 r per hour to 0.37 r per hour, and is now 0.86 r per hour, giving about 17 r per day (dose measured in air). Both rooms are lead insulated to insure adequate protection. A special meter in the hallway outside of the treatment rooms indicates the operating factors of the unit and records the hours of treatment, thus insuring uniform treatment and maximum safety. To accommodate nursing and medical care of the patient, diet hours and visiting, the unit operates twenty or twenty-one hours for each twenty-four hour period. With the present intensity factor of 0.86 r per hour, a dose of 100 r requires about six days of treatment.

For convenience of discussion, the cases have been grouped according to the pathological picture. Every case had an exact diagnosis determined either by biopsy, blood count (the leukemias and polycythemia), or roentgenographic examination plus other laboratory examinations when required. Numerous cases with questionable or equivocal diagnoses were discarded as unsatisfactory for exact analysis. About 10 per cent of the patients dying of their disease were necropsied. Any patient lost to follow-up was counted as dead at the date of last clinic visit.

An analysis according to diseases follows.

HODGKIN'S DISEASE

There were 94 unselected cases of Hodgkin's disease treated by localized therapy plus total body irradiation. This group includes a general cross section of all cases treated. The patients presented on admission disease varying from one or two node-bearing regions to generalized adenopathy of long duration. Recently, Slaughter and Craver[29] proved, from a study of a series of 265 cases of Hodgkin's disease, that one in every six patients is admitted with advanced terminal disease and dies within a period of six months or less after beginning therapy. This proportion of advanced cases is included in this series.

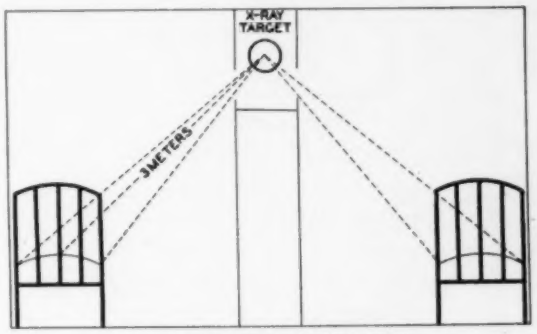

FIG. 1

It is our policy to evaluate each case of Hodgkin's disease according to the extent of involvement before any radiotherapy is begun. If early disease involving one or two node-bearing areas is present, an attempt is made to sterilize these areas using a fractionated dose of 2,000 to 2,400 r of high voltage therapy. With more generalized disease, a palliative dose varying from 400 to 1,200 r is applied to the areas showing the maximum involvement without any attempt at complete obliteration of the disease. From the beginning it was not deemed wise to select a group of patients to be treated by Heublein therapy alone, although it was recognized that such a plan would have simplified the evaluation of this method. The cumulative experience in a large group of Hodgkin's cases treated over a ten year period supports the fact that total body irradiation alone is usually insufficient to produce complete remission of the disease. Its greatest usefulness is as an adjunct to local high voltage roentgen therapy. This statement is borne out in the following analyses of survival rates and end-results.

The cases presented here have been analyzed for survival following onset of their disease and survival following onset of therapy at the Memorial Hospital (Chart 1). Nathanson and Welch[21] in a group of cases treated by irradiation, and Minot and Isaacs[18] in a group of untreated cases, computed survival rates from onset of the disease. As seen from Chart 1, the five year survival rate in untreated Hodgkin's disease is 12 per cent. In the 184 treated cases reported by Nathanson and Welch, the five year survival rate was 23 per cent. In our series of treated cases, 32 per cent of all cases survived five years from onset of disease; an increase in survival rate of 40 per cent. The median survival is forty-four months, an increase of fourteen months over that of the treated group reported by Nathanson and Welch,[21] and an increase of twenty-five months over the median of the untreated cases. The average duration of the disease of the 75 patients dead in this group was 44.4 months, an increase in survival of twenty-five months over untreated cases and an increase of fourteen months over those treated by localized irradiation (Nathanson and Welch). The average survival in this series for the 19 living patients is ninety-four months, and the median survival is 102.5 months.

In order to compare accurately the results of treatment of Hodgkin's disease in various cancer centers, it is essential to compute the proportion of early and late cases treated. It is obvious that the greater the number of early cases, the more favorable will be the survival rates following treatment. It is to be emphasized again that one-sixth of all patients with Hodgkin's disease admitted to the Memorial Hospital present advanced disease and die within a period of six months. Dated from the onset of complaints, the duration of the disease in this series before admission is 0 to three months in 20 per cent, three to six months in 25 per cent, six to twelve months in 32 per cent, one to two years in 11 per cent, two to three years in 8 per cent and over three years in 3 per cent. It is probable that as a group, the

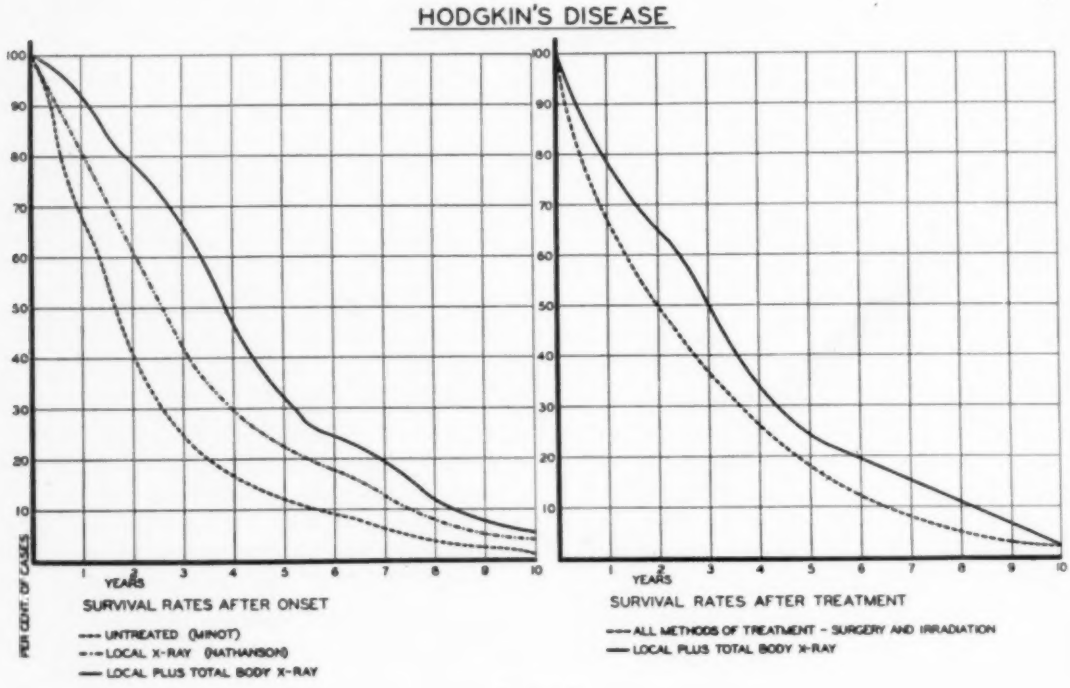

CHART I

cases of Hodgkin's disease admitted to Memorial Hospital are more advanced than those usually seen in a general hospital because one-quarter of the cases have previously been treated in other hospitals. In this series of patients, 23 per cent had received previous roentgen irradiation in other hospitals. Only 13 of the 94 cases presented disease localized to one or two node-bearing areas. Therefore, this series does not represent a select group of early favorable cases but rather a uniform group with mainly generalized or late disease. The improved survival rates following therapy at Memorial Hospital are therefore particularly significant.

The increase in survival rates following therapy is striking as seen in Chart 1. The average survival after combined radiation therapy at the Memorial Hospital is forty-two months; for all cases irrespective of the type of treatment, the average is thirty-four months. In the Heublein series, the median survival is thirty-six months following the primary roentgen therapy, and the five year survival rate is 24 per cent. This five year figure is an improvement of 6.2 per cent over the series of all Hodgkin's cases treated. Since survival rates from other clinics are reported from date of onset of disease, they are not comparable. Even so, in Nathanson's series the median survival rate is thirty months, six months less than our survival rate dated from the onset of treatment. Since the total body irradiation was administered at varying periods, both early and late, during the course of treatment, no deductions can be drawn from the rate of twenty-six months' average survival following its institution.

It scarcely seems justifiable to speak of five year cure rates in Hodgkin's disease, for, among a group of 100 untreated patients, 12 will be living at five years, but of these, 84 per cent will die of their disease during the next five years. The survival curve at ten years for the treated cases is almost identical with that of the untreated patients. One must therefore be wary of accepting any case as "cured" even though survival has been as long as ten years. In this series of 19 survivals (Table 1), 7 were living five years or more with no active disease during the last five years.

TABLE I

END-RESULTS IN HODGKIN'S DISEASE TREATED BY LOCAL ROENTGEN IRRADIATION PLUS TOTAL BODY IRRADIATION

Total number of cases	94
Number of patients dead	75 (80%)
Number of patients living	19 (20%)
Number living less than 5 years	7 (7.4%)
Number living 5 years or more with active disease	4 (4.2%)
Number living 5 years or more with no recent active disease	1 (1%)
Number living 5 years or more with no disease for 5 years or more	7 (7.4%)

It was of some interest to estimate the rôle played by Heublein therapy in the 7 cases "apparently cured." Five of these had early disease with only one or two node areas involved, 2 had mediastinal involvement and 2 had received neck dissections in other hospitals for early disease but showed recurrence shortly thereafter, and then received localized roentgen therapy to the nodes followed immediately by total body irradiation. Thus, we see that these patients had early disease and received obliterative therapy by surgery, roentgen rays, or both, followed by generalized irradiation. Throughout the whole group, response to treatment, as measured by survival and freedom from symptoms, was greatest where the disease was first well controlled by local therapy and this was followed immediately by generalized irradiation.

The 94 patients in this series received a total of 110 Heublein treatments. The cases were further analyzed for the exact extent of their response to treatment. It was found that 73 per cent showed improvement in symptoms and general physical condition; 27 per cent were unimproved. In 59 per cent, the demonstrable signs of disease were either reduced or held in check; whereas, in 41 per cent, the disease progressed in spite of therapy. In 25 per cent of the cases, improvement lasted six months or longer. Several facts stand out: (1) in the few cases

where total body irradiation alone was used as primary therapy, the initial response was good, but usually within a short period of time localized therapy was required to combat the recurrent or residual adenopathy; (2) the greater the amount of previous roentgen therapy, the poorer the response to Heublein therapy; (3) terminal cases of Hodgkin's disease showed no improvement from this therapy; (4) patients who received several Heublein treatments showed, in general, a diminishing degree of improvement with successive treatments; (5) maximum improvement from this therapy is seen where local disease is first controlled by local therapy.

The constitutional improvement which followed total body irradiation in 73 per cent of the cases was often striking. Many of these patients presented on admission, weakness, weight loss, malnutrition, anemia, and incapacitating malaise or pain which prevented them from carrying out any work. Gain in weight, loss of fever, improvement in nutrition and strength, and a general feeling of well-being usually followed therapy and, if the disease were well controlled, this improvement lasted from six months up to several years. If the disease was not controlled previous to the Heublein therapy, the resulting improvement was usually less marked and of short duration. For total body doses of 75 to 225 r, there appeared to be no definite correlation between the size of the dose and the degree of improvement. In a few cases, usually because of the advanced stage of the disease or the debility of the patient, a small dose of 25 r was given and was followed uniformly by little or no response.

The unfavorable constitutional reactions to total body irradiation in doses of 100 to 300 r are usually mild, transient, and present in about 20 per cent of the cases. It is our policy to interrupt Heublein therapy at any time the patient shows signs of developing marked gastrointestinal disturbance, weakness, apathy, fever, purpura, or unfavorable blood changes. Mild nausea, anorexia, or weakness was present in less than 20 per cent of the cases and cleared rapidly as the therapy was discontinued. In 2 cases with advanced disease, bronchopneumonia developed during the course of treatment but the therapy appeared to be a minor factor in its etiology.

The blood changes in Hodgkin's disease following total body irradiation are characteristic of the response of any human exposed to irradiation of sufficient intensity and duration over a large body area. In many cases, and lasting from a few hours up to several days, there is a transient increase of both red and white cells, suggesting a definite increase of hematopoiesis in response to the initial small amount of irradiation. In evaluating changes in the blood picture, this initial response was discounted entirely. The maximum changes were seen usually at the end of two or three weeks and persisted for several months. In a few cases, leukopenic reactions appeared first two or three months following the therapy. Some of these leukopenias can be attributed to the activity of the disease since they disappeared following further administration of small doses of local or generalized roentgen irradiation. However, for the purpose of these statistics, all such unfavorable reactions were attributed to the therapy.

The changes in the red cell count and hemoglobin were in general irregular and varied from 5 to 10 per cent above or below pretreatment levels. In 23 per cent of the cases, neutropenia followed therapy, and in 11 per cent, it was present at the onset of the treatment. Of the latter group, Heublein treatment was followed by improvement in 7, no change in 2, and further increase in the leukopenia in 2. In one-quarter of the cases, treatment was followed by a decrease of leukocytes of 2,000 to 10,000 without the development of leukopenia; in this group, the average fall was between 3,000 and 4,000 cells. In more than 40 per cent, there was no change or a slight increase in the white cell count.

The reaction of the blood to Heublein therapy depends on several factors: (1) the

general condition of the patient and activity of the disease; (2) the amount of previous irradiation; (3) the blood levels at the onset of therapy, and (4) the radiation dosage. It was apparent in numerous cases in which the general resistance of the patient was poor, or in which large amounts of radiation had been previously used, that unfavorable blood changes occurred, even after relatively small doses of 50 to 75 r. On the other hand, when factors (1), (2), and (3) were favorable, doses up to 300 r often resulted in an improvement of red cell count and hemoglobin without any marked shift in white cell count. When leukopenia is present in a patient with active generalized disease who has not received recent large doses of roentgen rays, Heublein therapy is usually followed by improvement of the leukopenia as seen in 7 of 11 cases in this series. Complete recovery from the leukopenia followed therapy within a period of several days to three weeks. Two of the 7 cases received each one transfusion during the period of recovery from the leukopenia. Because of the increased risk of agranulocytosis and serious complicating infections, total body irradiation is to be avoided in those cases in which leukopenia can be attributed to recent local irradiation.

LYMPHOSARCOMA

There were 30 cases of lymphosarcoma in this series, in 19 of which the pathologic diagnosis was reticulum-cell lymphosarcoma, and in 11 cases lymphosarcoma.* In 1 case the primary disease was found in the tonsil. Lymphosarcoma is recognized as a highly malignant lymphomatoid disease which, in its early stages, may involve one or two foci, but which, in the average patient, is seen as a generalized involvement of lymphoid structures. In the few reported cases of cure, the disease has been attacked radically in its early stages with but one or two areas involved. The average duration of symptoms before admission for treatment is twelve to thirteen months. In a survey of this disease in 1937, Jackson[15] concluded that patients with lymphosarcoma rarely survive the three year period and never the ten; reticulum-cell lymphosarcoma appears to be somewhat less malignant but also rarely permits survival beyond the ten year period. From a series of 196 cases at the Memorial Hospital, Sugarbaker and Craver[32] showed that the average duration of life in this disease was twenty-one months. Since lymphosarcoma is often a radiosensitive tumor and tends to widespread involvement, it would seem to be well adapted for Heublein therapy. However, in the treatment of this disease, as in Hodgkin's disease, our plan of therapy aims to bring the disease under control with local irradiation. Total body irradiation is reserved for those cases with generalizing or advanced disease, so that the end-results must be interpreted in the light of cases with a poor immediate prognosis. In no case with early disease did the patient receive Heublein therapy as primary treatment. In all 5 cases in which patients received this therapy primarily, the disease was so advanced and generalized that local irradiation was out of the question.

Table II

END-RESULTS IN LYMPHOSARCOMA TREATED BY LOCAL ROENTGEN IRRADIATION PLUS TOTAL BODY IRRADIATION

Total number of cases	30
Number dead of lymphosarcoma	27 (90%)
Number dead of other causes without recurrence	1 (3%)
Number living with active disease	0
Number living and well with no disease	2 (7%)
Patients surviving 5 or more years after treatment	4 (13%)
Patients dying of disease or living with active disease after 5 years	2 (6.5%)
Patients living and well with no active disease after 5 years*	2 (6.5%)

* Does not indicate that patient was continuously free of disease for 5 years but rather no active disease for at least 1 year.

In the evaluation of the end-results in this disease with such a bad outlook, one cannot measure the efficacy of the treatment by cure rates, for, as seen in Chart II,

* The term "lymphosarcoma" as a report on a biopsy means that the pathologist can recognize the disease as lymphosarcoma, while unable to classify it as belonging either to the reticulum-cell type or to the malignant lymphocytoma group.

there is no difference in ten year survival rates for treated and untreated cases. However, it is apparent from this graph that local irradiation plus total body therapy prolongs life up to five years in a greater number of cases. Thus at four years after the onset of the disease, twice as many patients are living as among the group treated by local roentgen irradiation alone, but at the six year period, there is no appreciable difference in the number surviving. The improvement in the survival rates up to five years is further demonstrated in average survival rates. For lymphosarcoma treated by all methods, the average survival after therapy is 11.8 months; for a group of 96 cases treated by local irradiation alone it is 12.4 months; and for this group of 30 cases, it is 24.2 months. Although there is an inevitable statistical error because of the small number of cases, this end-result for a group of unselected patients with generalized lymphosarcoma appears to indicate a significant trend. The five year survival rate for cases treated by all types of irradiation is 7.4 per cent; by local irradiation alone, 15 per cent, and by local roentgen irradiation and Heublein therapy, 11 per cent. If compensation is made for the advanced state of the disease in many of the latter group, the five year end-results compare favorably. The average survival after total body irradiation was 15.1 months. In this series the average duration of life from onset of the disease was 37.8 months, and there were no ten year survivals.

The average dose used in these cases treated with total body irradiation was 100 to 125 r and 37 such treatments were applied. On dividing the cases into two groups according to survival greater or less than twelve months, it was apparent that the size of the dose had no influence on the end-results. The average dose for 13 patients surviving more than one year was 120 r, and for 17 patients surviving less than a year, 130 r. In the former group, 4 of 13 cases had early disease and in the latter, 1 in the 17. However, the stage of the disease is not a greatly significant factor in relation to survival rates, for Sugarbaker and Craver demonstrated a difference of only

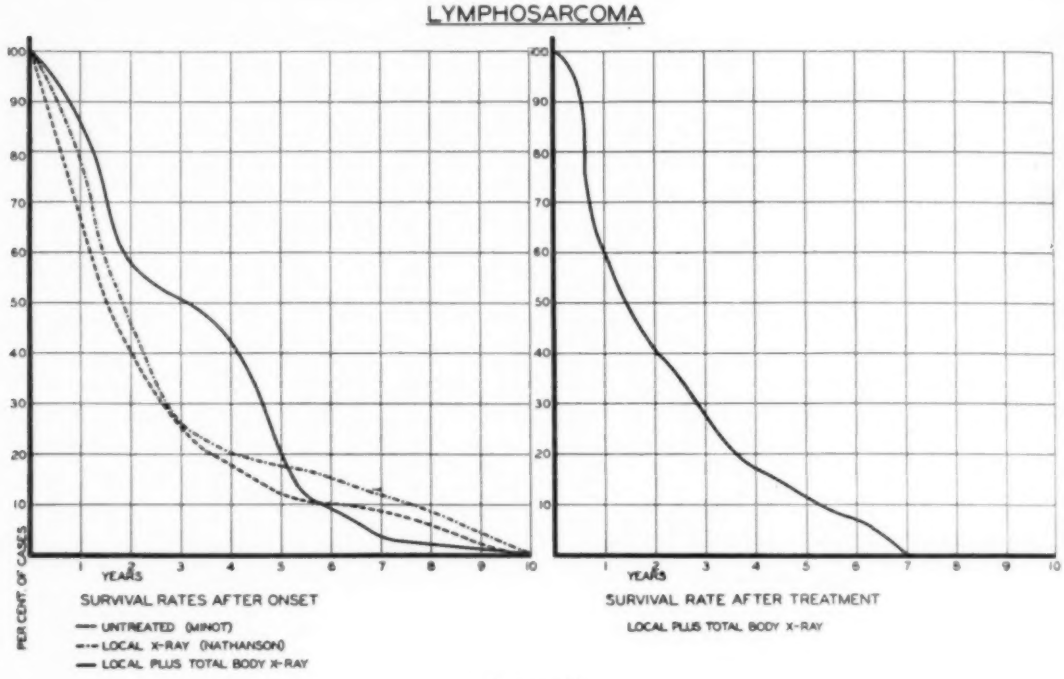

CHART II

one month for early and late disease.

In evaluating any therapy, one is not justified in examining only survival and cure rates without considering the effect of such therapy on the patient. Total body irradiation not only improved the health and well-being of the patients, but also reduced the amount and frequency of treatment in many cases. In 75 per cent of the patients there was both objective and subjective betterment with reduction in the amount of the disease, but in no case did complete regression of palpable disease follow this therapy. Here, too, the best results appeared in cases in which the disease was controlled by local irradiation followed by total body irradiation. The duration of this improvement varied from one month to three years, and in 8 cases following this therapy, there were no appreciable symptoms requiring any therapy for a period of one year or more. In 25 per cent of the cases there was no symptomatic improvement and in these the course of the disease was progressive.

One of us had the impression that following total body irradiation the interval between treatments by local irradiation was prolonged and this was borne out on analysis. The average interval between such treatments before total body irradiation was 6.2 months and following it, 9.7 months. Since local irradiation was applied only in areas showing active disease and was not used in a prophylactic way, this is further evidence that total body irradiation produces a definite growth restraint of lymphosarcoma.

Six of the patients showed mild transient distress from nausea or vomiting, and weakness during the total body treatment. One patient, with massive mediastinal lymphosarcoma, developed moderate dyspnea with pulmonary embarrassment, and one woman, aged thirty-four, following one treatment of 300 r, developed a permanent cessation of menses.

The blood changes in lymphosarcoma after total body irradiation follow a general trend for all cases of lymphomatoid disease except the leukemias. In about one-quarter of the cases, there was a rise in hemoglobin of 5 to 10 per cent, one-third showed no change and more than one-third showed a decrease of 5 to 10 per cent. These changes appeared usually at the completion of therapy but in a few cases were delayed for one to three months. In one-third of the cases following total body irradiation, a neutropenia developed. In 8 of the patients, neutropenia was present before the beginning of total body irradiation, usually as a result of previous local irradiation. In 6 of these cases, the further irradiation produced an increase in the amount of neutropenia, while in 2 there followed an improvement. More than one-third of the patients showed no change or a fall of leukocytes of less than 5,000 without the development of leukopenia. In most of the cases in which leukopenia developed, it was usually transient. However, when it was present before treatment and increased in severity, there was no response to repeated transfusions, and the leukopenia was often prolonged. Although there were no cases in which leukopenia was the precipitating cause of death, in 4 cases it persisted over several months up to the time of death, and appeared to be a contributing factor in lowering the patient's resistance to the disease and to terminal infection. From this it can be deduced that the blood picture is often improved by this therapy; leukopenic shifts are to be avoided if possible, and if leukopenia is present, the chances are that general body irradiation may produce harmful reactions.

LYMPHATIC LEUKEMIA

There were 76 patients with lymphatic leukemia treated by total body irradiation. Of these, 4 could be classified as having an acute form of the disease and all died within three months or less. Of the remainder, about 20 per cent ran a subacute course, dying within a period of twelve months, but are classified as chronic. Some authors classify as acute all those patients surviving one year or less and thereby are able to cite higher survival rates for chronic lymphatic

leukemia. This is scarcely justifiable for any critical evaluation of therapy. This series, therefore, represents a group of 72 cases of unselected chronic and subacute lymphatic leukemia with blood pictures varying from a typical leukemia to an aleukemia.

In the typical case at the Memorial Hospital, the plan for treatment of this disease is to irradiate first the external lymphadenopathy, using small doses of 100 to 300 r of high voltage roentgen therapy to each area. With a satisfactory response to this initial therapy and evidence of incomplete remission, the course may be repeated or similar treatment may be given through three portals anteriorly and posteriorly along the midline axis of the trunk. Liver and spleen are irradiated, depending on the amount of pathologic involvement. Total body irradiation may be used as primary treatment or as a supplement to local irradiation.

In this series, 7 patients are living, and 69 are dead. Of the 4 cases of acute lymphatic leukemia, each received from 10 to 25 r of total body irradiation without any demonstrable improvement or evidence of retardation of the disease. Similar dosage has been demonstrated to produce some favorable response in chronic leukemia. Acute lymphatic leukemia can be dismissed with the conclusion that general body irradiation, just as other methods of irradiation, does not alter its course or prognosis. In the group of chronic leukemias, all of the 7 living patients surviving from one year up to nine years after treatment, have low grade active disease. Although a few survive up to ten or twelve years, our findings coincide with those of others that there are none cured.

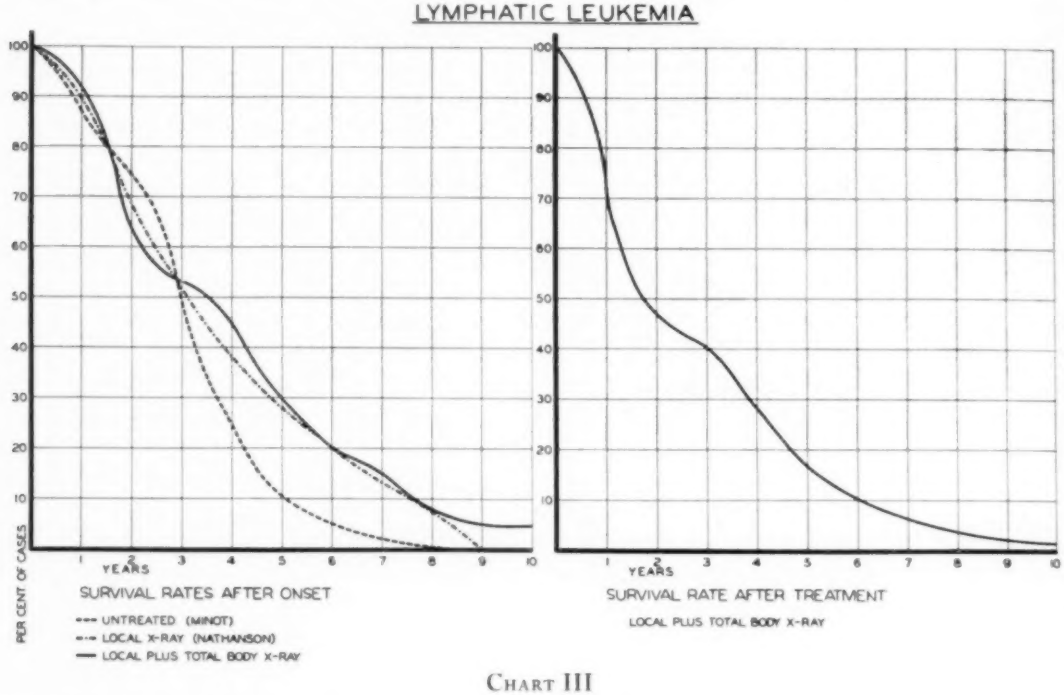

CHART III

Our experience has been that irradiation has a minimal effect in prolonging the life of these patients. As seen in Chart III, this is equally true for those treated by local high voltage roentgen irradiation and for our series of cases treated by additional total body therapy. Up to three years, there is no difference in the numbers of treated and untreated surviving. Past three years, the survival period is prolonged in the irradiated group, but at the end of ten or twelve years, whether treated or not, with rare exceptions all patients are dead. There

is a significant improvement in survival rates for those patients treated by total body irradiation over those treated by local irradiation alone. The average duration of life from onset of symptoms in untreated cases is 3.0 years; for a group treated by local high voltage roentgen therapy, 3.5 years and for our series treated by local roentgen irradiation plus total body irradiation, 4.0 years. Of Minot and Isaacs'[18] group of 60 irradiated patients, 42 per cent lived from two to four years, and 12 per cent more than five years and up to ten years *after onset*. In our series there were 14.4 per cent surviving five years or more *after treatment* as compared with 9 per cent reported by Craver[6] for all cases of lymphatic leukemia treated at the Memorial Hospital. The average survival after treatment in this series was 30.8 months.

It is generally recognized that although irradiation does not materially prolong life in chronic lymphatic leukemia, the period of useful life is significantly increased, especially when the irradiation is applied during the earlier stages of the disease. Total body irradiation is effective in inducing remissions, in maintaining a considerable number of patients symptom-free over variable periods of time, and in prolonging the duration of efficient life in a greater number of cases.

In 24 cases, general body irradiation was used as primary treatment and could therefore be evaluated *per se* without influence of local roentgen irradiation. In one-third of these, total body irradiation alone was employed throughout the course of treatment, usually because of the advanced stage of the disease. These patients received 38 such treatments, as many as 4 per patient. The results are tabulated according to response; satisfactory remissions in 70 per cent, partial remission in 5 per cent, and no remission in 25 per cent (most of the advanced cases were in this latter group). Sizable reduction in the amount of palpable disease, lymph nodes, spleen or liver occurred in 55 per cent, slight reduction in 10 per cent, and no demonstrable improvement in 35 per cent. The length of remissions obtained varied from two to fifteen months; the average was about five months. This series clearly demonstrated also that patients who had previously received large amounts of local high voltage roentgen therapy showed no favorable response to total body irradiation.

Significantly, of the 11 patients surviving five years or longer after treatment, all responded uniformly well to either local or general roentgen irradiation, and remissions were of longer duration. The longest remission of twenty-four months occurred in a patient who responded well to local irradiation over the external lymph node areas followed shortly by total body therapy. In only one of these cases, total body therapy was applied first during the terminal stage of the disease after many previous courses of local roentgen irradiation.

It was felt that there was some correlation between the sequence of application of local with total body irradiation and the survival period. The cases were therefore divided into three groups: (1) those receiving only Heublein therapy, mostly weak patients with far advanced disease; (2) those receiving one or two general body treatments followed by local irradiation; (3) those who received local irradiation staggered with one or several courses of total body irradiation. Only the cases in groups (2) and (3) were comparable. The most significant variable factor to be excluded in evaluating survival period after treatment of chronic leukemia is duration of disease before treatment.

RESULTS

Group (1)—10 cases (average duration before treatment was 26 months) had survival of 8.4 months after treatment.

Group (2)—19 cases (average duration before treatment was 15 months) had survival of 19.0 months after treatment.

Group (3)—34 cases (average duration before treatment was 14 months) had survival of 40.0 months after treatment.

Although the number of cases is too few to warrant final conclusions, it shows a defi-

nite trend, which, in the face of cumulative experience with other diseases, supports the thesis that survival is improved when the disease is first controlled by local roentgen irradiation followed by total body irradiation for growth restraint.

From this it may be concluded that, given a patient with chronic lymphatic leukemia, the best results are obtained when the patient is treated early in the course of his disease, first by deep roentgen therapy for maximal control followed by general body irradiation. By such a regimen, the duration of efficient life will be appreciably prolonged.

The blood changes in chronic lymphatic leukemia following total body irradiation fairly well demonstrate the efficacy as well as the dangers of this therapy in the leukemias. The dosage used varied from 25 to 200 r, but the average totalled 75 or 100 r. It is our policy to employ small doses of 25 to 50 r in cases in which there appears to be danger of a leukopenic reaction. One-third of the patients showed an increase of hemoglobin level of 5 to 15 per cent, 23 per cent showed no change, and 43 per cent presented a decrease of 5 to 15 per cent. The maximum changes in hemoglobin and red cell counts occur usually during the treatment but may be delayed for four to six weeks. Numerous cases showed an increase of anemia during irradiation but following treatment the hemoglobin slowly increased during the following three months. Since changes up to six months following treatment were recorded, it is apparent that some of the loss of hemoglobin can be in part attributed to the natural course of the disease.

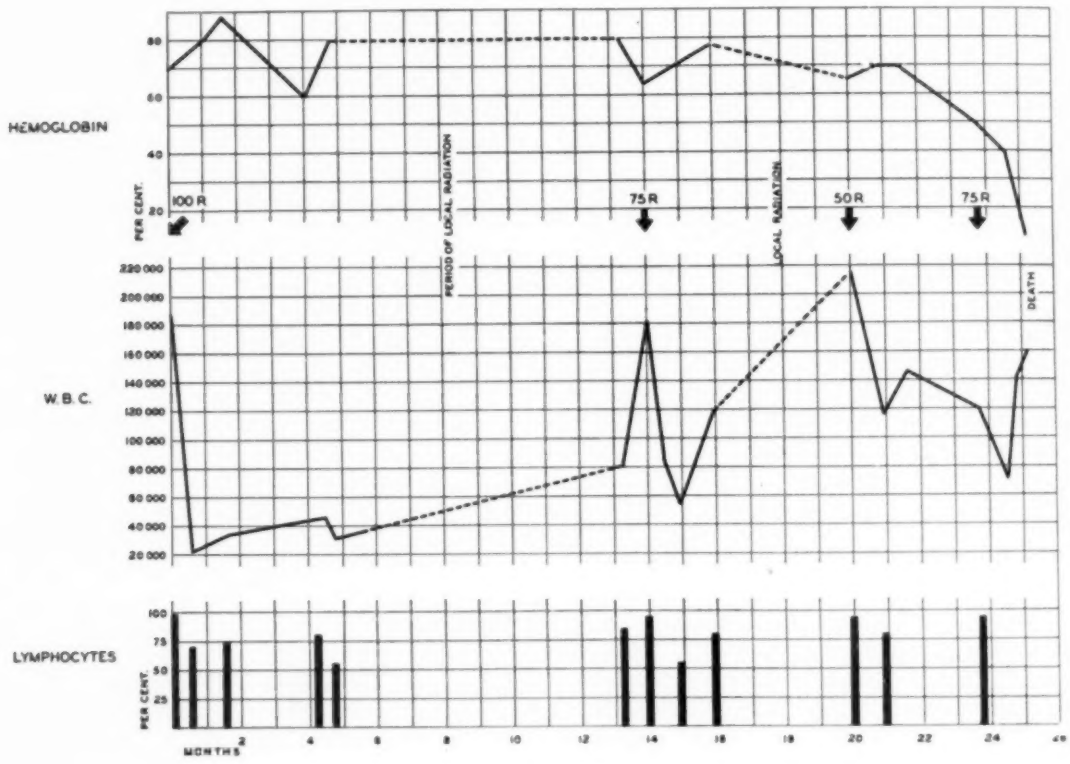

CHART IV

The changes in the white cell counts following Heublein therapy proved interesting because of their wide divergence with shifts of 5,000 to 350,000 in 70 per cent of the cases. For the dosages employed, there was no constant relation between the dosage

and the decrease in white cells. The more acute phases of chronic leukemia with very high white counts presented the maximum decrease, often of 100 to 200 thousand cells, whereas those cases in an aleukemic or subleukemic phase usually showed decrease of 1,000 to 2,000 cells. Case 1 illustrates the hematologic course of one patient with active chronic lymphatic leukemia in response to total body irradiation and demonstrates that the level of the white cell count before treatment largely determines the amount of the decrease (Chart IV). Following the first dose of 100 r the white cells decreased by 175,000 within one week and the patient was well. Four months later, with onset of recurring complaints, the white cell count was elevated to 46,000 and 50 r given at this time produced a decrease of only 16,000 cells. Subsequently acute exacerbations of the disease attended by very high counts responded to doses of 50 and 75 r by prompt and marked decreases of the white cell level. On the other hand, a representative patient with active chronic aleukemic leukemia (Case 11, Chart V) showed a white count of 4,800 with 60 per cent lymphocytes on admission and following 100 r of total body irradiation, the count remained for six months at 5,000 to 6,000 cells with 25 per cent lymphocytes. In contrast, another patient with active leukemia, showing a white count of 19,000 before treatment, developed a leukopenia of 4,000 cells at the completion of 25 r. The shifts in the white cell levels are often rapid, unpredictable, and must be followed daily during the period of treatment in order to guard against overdosage.

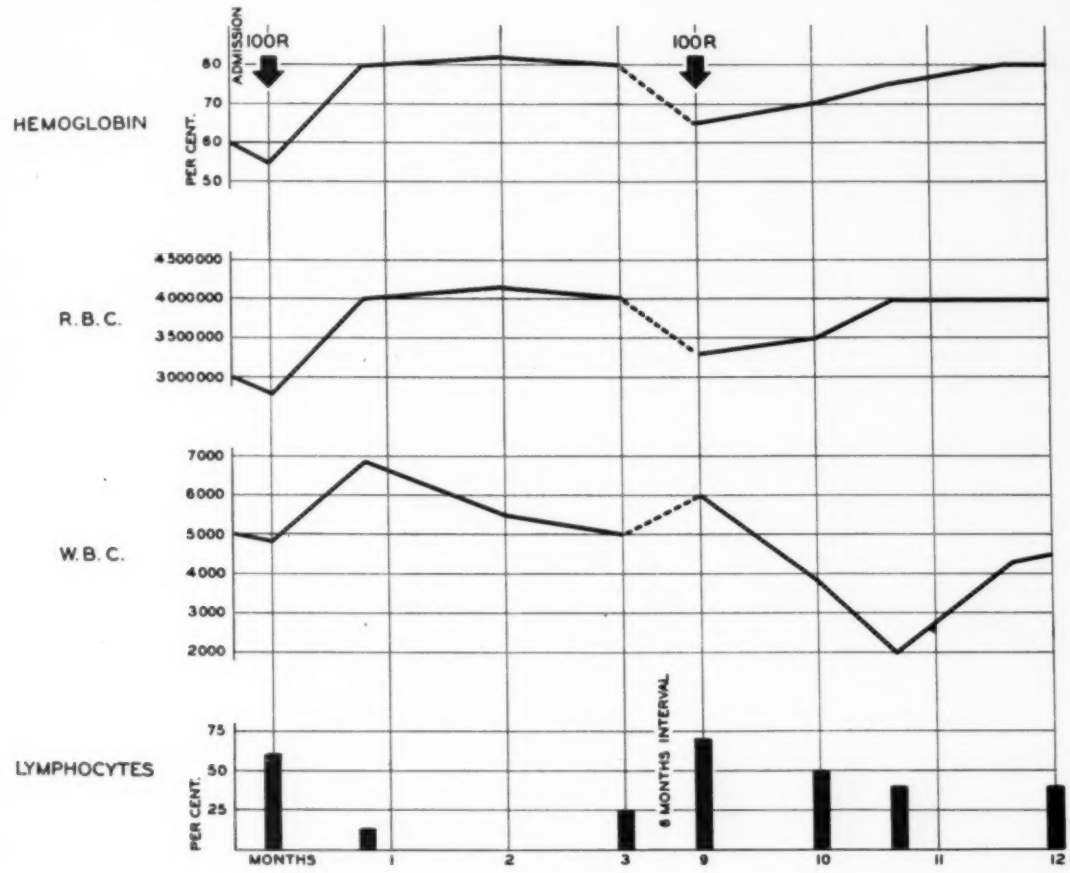

Chart V

In 4 cases there were no changes in cell

count and in 6 cases there was an increase. Half of the latter were cases with counts between 5,000 and 10,000 who improved symptomatically. In 9 cases, therapy was complicated by the development of leukopenia. For the greater part, this occurred when the counts before treatment were low. The counts before treatment in the cases developing leukopenia follow: 5,000 to 10,000—7 cases; 10,000 to 20,000—2 cases; 20,000 to 50,000—1 case, and over 50,000—2 cases. In 8 instances, the leukopenia was transient, lasting from a few weeks to three months. In 4 cases leukopenia with anemia complicated by a terminal hemorrhagic diathesis was progressive and death ensued within one to four months.

In 5 cases, leukopenia was present at the beginning of the therapy and in these, six treatments were followed by further temporary decreases of white cell count, two treatments were followed by no change and four treatments produced permanent increase in the counts, along with symptomatic improvement. Thus, leukopenia *per se* is no contraindication to roentgen therapy and may be improved especially in those cases with aleukemic active disease.

Of all the cases following total body irradiation, 87 per cent showed a decrease of white cells and 13 per cent showed no change or a slight increase. The maximum reduction in the white cell counts occurred within one to two weeks after treatment but in a few cases the fall was prolonged up to two or three months. The improvement in the blood picture is sustained on the average for three months and in the exceptional case for as long as five to six months.

From the blood studies, it was apparent that the amount of decrease in the white cell count depends not only on the dose of radiation but also on the level of the count before the total body irradiation. Cases showing an aleukemic phase with counts of 20,000 or less frequently developed a leukopenia. On the other hand, cases with low grade active leukemia with counts up to 20,000 or 30,000 showed a minimal decrease in white cells, but often striking clinical improvement. The degree of clinical improvement sometimes does not parallel the change in the blood picture. There may be a marked decrease in white cells with no change in nodes and spleen or there may be a minimal change in leukocytes with marked regression of nodes and spleen. Thus, it is clear that the blood picture *per se* is not always a reliable index to the response to therapy.

MYELOGENOUS LEUKEMIA

Twelve patients having myelogenous leukemia received total body irradiation. All are dead. One patient had the acute type and although temporarily improved by this therapy, developed shortly thereafter a hemorrhagic diathesis and died two months after treatment. The average duration of the disease in the 11 chronic cases was 28.5 months after onset, and 18.6 months after treatment. Although a few cases of this disease are known to show survival for as long as ten to fifteen years, the longest survival after treatment in this group was forty months.

At the Memorial Hospital, the plan of treatment in the typical case is deep roentgen therapy through large portals anteriorly and posteriorly over the spleen in doses of 25 to 100 r for a total of 200 to 300 r to each of two portals. Care is taken to avoid overdosage and thus avoid acute toxic reactions. During the early course of the disease, size of spleen and leukocyte levels offer valuable guides to the efficacy of treatments or need for more therapy. Total body irradiation is usually reserved for later stages of the disease when the splenomegaly has been controlled by local roentgen therapy, but the blood shows marked leukemia. In this latter stage, remissions by any form of therapy are more difficult to produce and of shorter duration.

From the results, it may be seen that total body irradiation offers little or nothing over local irradiation by way of producing remissions or prolonging life. The 11 cases received eighteen total body treatments with an average dose of 50 r and up to 150 r

in 2 cases. Following thirteen treatments to 8 patients, remissions of from two to three months in duration were seen. Three patients received five treatments from which little or no improvement could be demonstrated. The same patients responded poorly to local roentgen irradiation directed to the spleen. In 2 patients for whom 150 r total body irradiation was used first, there resulted a partial and temporary reduction in the size of the spleen, but as a general rule, no regression of spleen or nodes was apparent. From this small group, the uniform results suggest that those cases received the most palliation in which total body irradiation was used either primarily in treatment or shortly after treatment by local irradiation. Contrary to the observation of Sgalitzer, advanced cases with spleens refractory to local irradiation showed no marked response to general body irradiation.

The improvement in the leukemic and anemic blood pictures following this therapy serves as a good index of the clinical improvement. In more than half of the cases, therapy resulted in a rise of red cells and hemoglobin of 5 to 10 per cent, and in one case, as much as 20 per cent. An effective reduction in the immature myelocytes in the blood is the rule. The extent of the decrease depended mainly on the blood level before treatment and the dosage, and varied from 10,000 to 225,000 cells. As a general rule, the higher the myelocyte count, the greater the decrease following therapy. In one case, therapy was followed by a progressive increase of the leukemia, and in another case, treatment effected no change in the leukocyte count.

We conclude that total body irradiation offers little or nothing over local roentgen therapy in the early treatment of this disease. Later, when the spleen becomes refractory to further local irradiation, total body therapy may produce some clinical improvement lasting for a few months, but no significant alteration in the length of the course of the disease. We feel certain that total body, as well as local roentgen therapy has proved a worthwhile method in improving the general health and constitution of these patients so that their useful life is definitely prolonged.

POLYCYTHEMIA VERA

Polycythemia vera represents a systemic disease in which there is an overgrowth of cells in the erythropoietic system, resulting in a marked increase in the number of circulating red cells, as well as an actual increase of blood volume. This disease may be complicated by a myeloid leukemia as was seen in 2 of the 5 cases being reported and is then called erythroleukemia. The aim of the treatment of this disease is to depress the erythropoietic elements of the bone marrow and thereby reduce the volume of circulating red cells and blood. The logical first sites for treatment are the marrow of the long bones, sternum, and vertebrae. It has been our experience that local high voltage roentgen therapy over these areas produces satisfactory lowering of the red counts with coincidental remission of symptoms for many months. The spleen is not irradiated unless erythroleukemia is present, or unless it becomes so large that distressing mechanical compression is present. Later, one or more courses of total body irradiation may be used. The results of local radiation therapy for polycythemia vera have previously been reviewed by Pack and Craver.[23]

The treatment of 5 patients who received total body irradiation is analyzed. The first 2 patients with associated myeloid leukemia died one and forty-four months after treatment. The duration of the disease after treatment in Cases III and IV was seventy-seven and 151 months. The fifth patient is living and is comparatively well seventy-three months after admission.

From an evaluation of the end-results of both local and total irradiation in this small group of cases, it is impossible to prove that either method is more effective than the other method. In general, the disease seems to remain under satisfactory control for long periods of time by both methods. In 4

cases, total body irradiation by doses of 50 to 100 r produced remissions lasting from three to fifteen months. In the first case of erythroleukemia, with diminishing response after two years of local irradiation of the spleen and bones, four total body treatments during the next two years gave gratifying palliation for periods as long as eleven months. Following treatments, the decrease in the red blood count varied from 1 to 4 million cells. The second patient with erythroleukemia untreated for five years failed to respond to a dose of 100 r and died as a result of severe gastric hemorrhage at the end of treatment. A third case of polycythemia treated by repeated courses of local therapy from 1928 to 1935 showed satisfactory palliation lasting three months after the first total body irradiation, but two subsequent treatments produced a minimal response. No significant changes in the red cell counts followed the treatments. Case IV, treated two years previously by a small dose of local irradiation, responded very well to 100 r of total irradiation. The red cells decreased from 10 million to 6.5 million and a remission of fifteen months followed, during which the patient returned to work and enjoyed good health. The fifth case has been treated by local irradiation of the bones, "spray therapy" to the chest and abdomen, total irradiation and phenyl-hydrazine, and each has seemed to produce a satisfactory response. Following total body irradiation, plethora was controlled for nine months.

Our results are encouraging but, due to the small number of cases treated, one cannot draw any conclusions for the superiority of total body irradiation. Reznikoff[24] treated 3 cases by total body irradiation and demonstrated greater responses than were seen after local therapy. It is interesting to note that Hunter,[14] using total body irradiation in 2 patients over a period of several months for total doses of 900 and 1,200 r, was able to effect in both cases complete remissions for three years, dated at the time of the report. He concluded that the superior results could be attributed to the profound depressant effect of roentgen rays administered in small doses over long periods of time. In the largest series of cases of erythremia reported, Sgalitzer[28] cited excellent results from total body irradiation in 42 of 44 cases and claimed that with recurrences one to five years later, he obtained equally good responses as in the first cycle of total body irradiation. We are unable to confirm the opinion that total irradiation in the late stages of the disease gives satisfactory remissions. Here, as in chronic leukemia, it is our impression that total body irradiation may offer its greatest effectiveness when used as a supplement to local irradiation.

MYCOSIS FUNGOIDES

Mycosis fungoides, a lymphomatoid disease primarily involving the skin with multiple discrete widespread nodules but also occasionally involving the lungs, bone and other viscera, responds best to treatment by low voltage roentgen therapy directed to the largest and most extensive areas of involvement. When the disease becomes generalized, one is forced to resort to generalized palliative therapy. Six of our cases, after many previous treatments by local irradiation, were given a trial of total body irradiation, in dosage varying from 50 to 150 r. In 4 of the cases, there was a favorable constitutional improvement with partial regression of the lesions. There was a complete subsidence of the lesions over the trunk and extremities for two months in one case; in another, relief of intense pruritus resulted. In all it was apparent, however, that any control attained by total body irradiation was of brief duration. Following therapy in 2 cases, there was no demonstrable improvement. It may be concluded from these few cases, that total body irradiation offers brief and incomplete palliation of the advanced generalized stage of this disease. Except for transient leukopenia in 2 cases, there were no complications resulting from total body irradiation.

MULTIPLE MYELOMA

Eleven cases of multiple myeloma were treated by total body irradiation. The diag-

nosis was made in all cases by the roentgenographic appearance of typical multiple lesions throughout the skeleton plus the appearance of Bence-Jones protein in the urine. In 7 of the cases, the diagnosis was proved by biopsy. The natural course of this disease averages about two years and an occasional patient will survive five years or longer. Pain due to the actual bone involvement or to the compression of the spinal cord or peripheral nerves is most pronounced in this disease. Early in the disease when few lesions are present, localized roentgen treatment to the involved bones produces satisfactory regression, but when the disease becomes generalized, total body irradiation may offer the greatest hope for palliation. Of the 11 cases reported, 6 received local irradiation before total body therapy, 2 received primarily total body therapy followed by local irradiation to areas giving rise to pain and 3 received total body irradiation as the only irradiation plus Coley's toxins. All cases presented generalized skeletal involvement at the time of the total body irradiation.

There were thirteen treatments given to the eleven cases with doses varying from 50 to 300 r depending on the general condition of the patient. The average dose was 200 r. Following nine treatments in 7 cases, there was definite improvement as demonstrated by gain in weight and partial or complete relief of pain for periods of three to eight months. In 2 cases, this improvement lasted but two weeks. Following four of the treatments, no symptomatic improvement could be elicited.

Although in some cases apparent arrest of the disease in a particular focus can be accomplished by local deep roentgen therapy, such local regression of lesions cannot be expected from the small doses realized with total body irradiation. However, in one of our cases, coincident with the marked relief of pain, there was moderate regression of the palpable metastatic masses over a three month period. A second patient with generalized skeletal involvement of plasma cell myeloma, proved by biopsy, and previously treated at another hospital by local irradiation, received only a long course of Coley's toxins, plus 250 r by total body irradiation. There was marked improvement of the patient, with roentgen evidence of excellent regression of the lesions. Five years after treatment here, a letter from his physician in Ireland where he was residing, stated that the patient was living and well with no complaints, and that recent roentgenograms of the skeleton showed no recurrent lesions. It is impossible to say what rôle total body irradiation played in the unusually successful outcome of this case. Another patient, following previous local irradiation of the bone lesions in other clinics, responded very well to total irradiation of 100 r, and for the first time in many months was able to lead an active, comfortable life. This favorable palliation, with gain of 10 pounds in weight, lasted for more than one year. In several cases this therapy appeared to hold the disease in check for periods of three to six months so that there was a definite prolongation of the interval before further therapy was needed. On reviewing the 4 cases surviving two to five years after treatment, it was evident that no constant plan of therapy produced these superior results. Although some cases naturally survive longer than others and the cases are too few to permit any certain conclusions, it is our opinion that total body irradiation may materially increase the survival period in this disease.

The anemia incident to progressive malignant disease is also seen in these patients. In line with the symptomatic and constitutional improvement realized by total body irradiation, one-half of the cases showed an increase of hemoglobin of 5 to 10 per cent. Transient leukopenias developed in one-third of the cases, following doses of 250 to 300 r, and in 3 cases in which leukopenia existed before therapy, there was a further decrease of white cells. No significant change in the white cell count was recorded in 4 cases.

Two cases of endothelial myeloma (Ewing's tumor) with advanced metastatic disease received palliative irradiation by total body treatments. One was symptomatically

greatly improved for three months, and partial regression of the palpable disease occurred temporarily. In the second case, treatment was followed by constitutional improvement with slight reduction in the large mass in the mandible lasting six weeks.

MISCELLANEOUS GROUP

In this group are 35 patients having advanced carcinoma or sarcoma with generalized metastases, most of whom received total body irradiation during the period of 1931 to 1933 when the method was first being evaluated. There were 13 cases of breast carcinoma, 5 of lung carcinoma, 4 of generalized melanoma, 3 of teratoma of the testis, 2 of carcinoma of the prostate, 3 of metastatic carcinoma, primary lesion undetermined, 2 of carcinoma of the kidney, and 1 each of carcinoma of the tonsil, carcinoma of the skin of the face, and carcinoma of the thymus. Except for transient relief of pain in a few cases, the results in these generalized carcinoma cases were discouraging. The reason for this is quickly apparent. Carcinomas are much more radioresistant than the lymphomatoid tumors, and by total body irradiation the dose cannot be nearly large enough to alter these tumors appreciably. Wherever carcinoma or sarcoma exists, a cancericidal dose to the tumor must be supplied in order to eradicate the disease, and any dose short of this is relatively ineffective. The largest doses administered by total body therapy fall far short of any cancericidal effect, so that the reason for the uniformly poor response in these cases is obvious. If total body irradiation can be of any value in cases of carcinoma or sarcoma, it must be applied as a prophylactic treatment supplementary to radical measures to eradicate localized disease. We can report no such group of cases, for the value of such therapy would be very questionable in the light of our previous experience. However, Mallet,[17] in a review of 300 advanced cancers with widespread metastases, claimed that he obtained marked palliation in most cases, and in some, almost complete regression of bone metastases with the use of teleroentgen therapy, dividing the body into four portals and avoiding treatments over the long bones. We are unable to find any other reports supporting Mallet's experience, but believe this method merits further trial. Radioactive phosphorus offers some as yet unevaluated hope of producing total body irradiation over a sufficient length of time to destroy or inhibit development of metastases of cancer, but here again it would seem that a cancer-lethal dose of the material will produce deleterious reactions in the bone marrow and general metabolism which may prove lethal to the patient.

DISCUSSION

The results of the treatment of 270 cases demonstrate that total body irradiation produces the greatest palliation in the group of lymphomatoid diseases and therefore its usefulness is largely restricted to this group of radiosensitive tumors. The diseases showing the greatest benefit from this therapy are chronic lymphatic and myelogenous leukemia, Hodgkin's disease, lymphosarcoma, and polycythemia vera. Less striking improvement was noted in mycosis fungoides and multiple myeloma. No appreciable improvement followed this therapy in the cases of generalized metastatic carcinoma or sarcoma.

Although in many European clinics total body irradiation has been abandoned in favor of teleroentgen therapy because of serious or fatal reactions, our experience shows that this method is safe when used judiciously, with the knowledge that excessive dosage produces a deleterious effect on the course of the disease as well as definite harmful reactions to the patient. In not more than 12 cases in this series was it felt that the patient had received an overdosage of irradiation, usually a combination of local high voltage roentgen therapy plus total body irradiation, which resulted in the weakening of general resistance to the disease or infections and so hastened death. There is no exact rule for the selection of

dosage. The aim of therapy is to obtain maximum improvement with minimal unfavorable reactions. In the determination of this dosage one must be guided by several factors: (1) age and general resistance of the patient, (2) the disease and its extent, (3) the amount of previous irradiation, (4) the blood cell levels previous to therapy, and (5) the response of the patient during treatment. Frequent blood counts during treatment are a necessary guide to the effect of therapy on hematopoiesis and resistance of the patient. In the radiosensitive leukemias, a maximum continuous dose of 100 to 150 r usually proved adequate; in Hodgkin's disease and lymphosarcoma doses of 100 up to 300 r were used. It is important to caution against acceptance of these doses as standards which may be adopted for every case in the several disease groups. The treatment of each case must be carefully evaluated beforehand and the dosage individualized. In order to evaluate the response of the patient and the disease to therapy numerous cases receive a small dose of 25 r and if a satisfactory response is obtained, therapy is renewed for an optimum dose. A dose of 300 r, given in one continuous course in ten days appears to be the upper limit of safe dosage for a patient in good health, but it is to be emphasized that in many patients, transient toxic reactions appear which preclude dosages higher than 50 or 100 r. Marked vomiting, weakness, apathy, fever, purpura, or hemorrhage and development of leukopenia, are the danger signals calling for discontinuance of total irradiation. In 20 per cent of the patients, mild gastrointestinal distress or weakness was apparent but usually cleared rapidly following completion of therapy.

Leukopenia resulting from depression of the bone marrow is the most important complication of total body irradiation and wherever possible should be avoided. Varying with the groups, it appeared in 10 to 30 per cent of our cases, and, if the dosage had not been excessive, was of brief duration. In the leukemias and lymphosarcomas, leukopenia usually was apparent at the end of treatment and in only a few cases was delayed up to two or three months. In Hodgkin's disease and the radioresistant tumors, leukopenia most often first appeared two to eight weeks following treatment and in a few cases was delayed for five to six months. In the latter, however, factors other than irradiation may have been responsible for the leukopenia.

In no case with a white count of greater than 5,000 before treatment did total body irradiation result in a permanent or fatal neutropenia. On the other hand, in 8 cases showing no response to localized therapy and with definite leukopenia, total body irradiation was followed by further decrease in white cell count which persisted in spite of active supportive therapy until death. It is to be emphasized, however, that leukopenia *per se* is not an absolute contraindication to this therapy, for in about half of the cases of lymphatic leukemia in a leukopenic phase, therapy was followed by a return of leukocytes to normal levels. In general, wherever leukopenia exists as a result of recent large doses of localized irradiation, increase in leukopenia may be expected from further roentgen therapy, but if leukopenia exists because of active disease, improvement in the blood picture following therapy may be anticipated.

The results in this series of cases indicate that total body irradiation produces the greatest benefit when combined with local high voltage roentgen therapy. As measured by symptomatic improvement or remission of the disease, survival rates after treatment, number of five year survivals and lengthening of the period of useful active life, those cases showed optimum benefit in which the disease was first controlled by local irradiation followed shortly by a course of total body therapy. The constitutional improvement is manifest by a sense of well-being, betterment of gastrointestinal function, weight gain, and increase of hemoglobin. It has been suggested that the improved end-results in these diseases following irradiation may depend to a con-

siderable extent on the improvement of the constitution of the patient. That total irradiation also exerts a definite effect on tumor growth-resistance is apparent not only by the longer intervals of freedom of disease in numerous cases, but also in the better survival rates following treatment. This is especially apparent in Hodgkin's disease and lymphosarcoma. In the advanced or radioresistant cases of lymphomatoid disease, the responses to total body therapy were minimal and so we are unable to confirm the impression of others that these cases respond more satisfactorily to total body irradiation. In cases of generalized carcinoma, total irradiation produced no significant improvement.

Numerous theories have been advanced to explain the beneficial effects of general body irradiation. All agree that this therapy exerts chiefly a constitutional effect so that the resistance of the patient to his disease is increased, but the exact mechanism remains obscure. That a definite biochemical change takes place in the blood elements following total irradiation has been proved recently[1] and this may be the first clue to defining the exact mechanism of irradiation effects on all cells.

SUMMARY

1. The results of treatment by total body irradiation of 235 cases of radiosensitive tumors, mostly lymphomatoid diseases, and 35 cases of radioresistant generalized carcinoma are reviewed.

2. The techniques of total body irradiation at Memorial Hospital and of teleroentgen therapy as used in other clinics are summarized.

3. The dangers, complications, and contraindications to total body irradiation are evaluated.

CONCLUSIONS

1. Total body irradiation has its greatest usefulness in the treatment of the lymphomatoid diseases. Little or no benefit follows its use in the treatment of generalized carcinoma or sarcoma.

2. The survival period as well as the period of active useful life is prolonged in patients with Hodgkin's disease, lymphosarcoma and chronic lymphatic leukemia treated by total body irradiation.

3. Favorable palliation followed the use of total body irradiation in small groups of patients with chronic myelogenous leukemia, polycythemia vera, mycosis fungoides, and multiple myeloma.

4. Acute lymphatic and myelogenous leukemia are not benefited by this therapy.

5. The most favorable results for the treatment of the lymphomatoid diseases were seen in those cases with early localized disease where obliterative therapy by local roentgen irradiation or surgery preceded total body irradiation.

6. Total body irradiation is a useful adjunct to local roentgen therapy in the treatment of the lymphomatoid diseases.

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RADIOTHERAPY OF CANCER OF THE ANTRUM*

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THE purpose of this paper is to present four malignant tumors of the antrum treated by external irradiation and also to discuss a new technique of radiotherapy known as the concentration method.

The methods which have been used in the treatment of cancer of the antrum are surgery, including electrocoagulation, combined surgery and irradiation, and external irradiation alone. Surgery has had its greatest success in the treatment of lesions arising in the lower half of the antrum. When cancer of the antrum arises in its upper half, it soon invades the orbit, the ethmoids and the pterygopalatine fossa. Complete surgical removal of such lesions is generally unsuccessful and, at best, highly mutilating. In spite of these difficulties, however, New and Cabot reported 40 per cent of their patients alive and without recurrence after operation.

The frequency of local recurrences following surgery has led to the use of combined surgery and irradiation. Thus, Hautant and Monod developed a combined procedure in which the lesion is removed surgically and radium tubes are placed in the operative wound. These authors report 17 extensive carcinomas of the maxillary sinus treated by combined radical surgical resection and local radium therapy. Five patients were alive two to eight years after treatment. The complications of this combined procedure are osteoradionecrosis, infection and hemorrhage. Öhngren reported 14 cases treated either by electrosurgery alone or combined with radium. The primary mortality was 11.4 per cent. Thirty-eight per cent were well after five years.

The best results of external irradiation alone in the treatment of cancer of the antrum are reported by del Regato from the Curie Institute of Paris. Ten patients with extensive, inoperable cancers of the antrum were treated by roentgen therapy alone. Four patients were well from five to fifteen years at the time of the report (1937). The total dose in these cases was between 6,000 and 7,000 r (measured on the skin) and given over a period of five to six weeks. Two fields were employed, one lateral and one anteroposterior; 200 kv. roentgen rays were used; and the daily dose varied from 200 to 250 r. The treatment resulted in the production of the usual "epidérmite" and "epithélite."

METHOD OF CONCENTRATION

The divided dose technique which is the method of roentgen treatment now generally used is given over periods varying between twenty and forty days. The daily doses vary between 350 and 150 r. The total dose to each field varies between 3,000 and 4,000 r. This method results in a cure of a certain percentage of cancers of the mouth, pharynx, larynx and accessory nasal sinuses, but the cures are limited to the more radiosensitive lesions.

Since 1938 the staff of the Chicago Tumor Institute has been studying different methods of roentgen and radium therapy in an effort to cure carcinoma of the mouth, pharynx, larynx and accessory sinuses which belong to the radioresistant group. The results of these studies were reported recently by Cutler who has applied the term "concentration method" to this new technique. The following is a description of the method:†

Large daily doses of roentgen rays or radium are used over short treatment periods (nine to twelve days). The total dose is sufficient to produce the characteristic "epithélite" in the mucous membrane and "epidérmite" of the skin. In selected cases the daily dose is increased as the size

* From the Chicago Tumor Institute.

† For complete details of the technique see Cutler.[1]

of the portal is diminished; 200, 400, and 500 kv. roentgen rays are used. (A parallel series of cases has been treated with gamma rays by means of the 10 gram radium bomb.) The intensity has varied from 3 r per minute to 10 r per minute. From one to three fields have been used for cancer of the mouth, pharynx, larynx and accessory sinuses, depending upon the site and extent of the lesion. This technique is in no way intended to be final and is still under study but it does represent what appears to be a definite advance in the external irradiation of the more radioresistant forms of cancer of the mouth, pharynx, larynx and nasal accessory sinuses.

REPORT OF CASES

Case 1. C. O., white, male, aged fifty, admitted May 17, 1938. Pain in right upper molar region for one month followed by recession of gum, loosening of teeth and swelling of right side of face. Examination disclosed an ulcerated lesion, 4 by 5 cm., which had partly destroyed the right upper alveolar ridge. In the center of the ulcer was a loose molar tooth. Externally there was a swelling over the right superior maxilla. There was no adenopathy. Roentgen examination disclosed marked clouding of the right antrum, widening of the lateral and inferior antral walls, and increase in density of the right upper alveolar ridge. The loose molar tooth was extracted and a biopsy taken from the edge of the ulcer. Histopathological examination disclosed epidermoid carcinoma with hornification and pearl formation. Roentgen

Fig. 1. Case 1. Squamous carcinoma of antrum before treatment.

Fig. 2. Case 1. Squamous carcinoma of antrum after treatment.

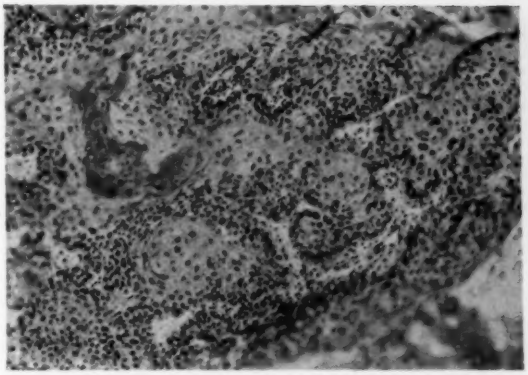

Fig. 3. Case 1. Photomicrograph showing squamous carcinoma.

Fig. 4. Case 1. Roentgenogram before treatment showing clouding of right antrum.

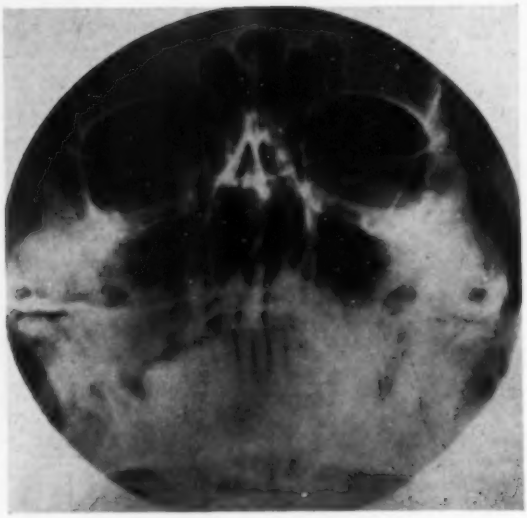

FIG. 5. Case I. Roentgenogram of right antrum after treatment.

therapy was administered as follows: Voltage, 400 kv.; filter, 6 mm. Cu; distance, 83 to 100 cm.; dose, 6,850 r (measured on the skin) in twelve days (16 sittings). One anterolateral portal was used. The daily dose varied between 300 and 800 r, and the fields were gradually diminished from 84 cm.^{2} to 10 cm.^{2}. The roentgen intensity varied from 4.3 r per minute to 7 r per minute. Both eyes were protected through the entire course of irradiation. An "epithélite" first appeared on the eighth day. The tumor began to show signs of regression on the eleventh day; the radiation reaction reached its height on the nineteenth day; a moist "epidérmite" developed on the twenty-second day and healed after eighteen days. The ulcerated lesion healed one month after the beginning of treatment. One year later, a cutaneous and subcutaneous nodule, 2 by 3 cm., developed in the right cheek over the zygomatic process in the center of the irradiated area. This was interpreted as a radiation effect in the subcutaneous tissues and it disappeared after four months under hot compresses. The patient is well three years and four months after treatment. There is slight atrophy of the skin over the treated area. There is no clinical or roentgenological evidence of disease.

CASE II. T. K., white, male, aged forty-seven, admitted August 28, 1939. Pain in left side of face and head for five months. Four months later a perforation appeared in the left upper alveolar ridge. A Caldwell-Luc operation was performed in July, 1939, and the biopsy showed squamous carcinoma. Examination disclosed a fullness of the left side of the face and slight proptosis of the left eyeball. An extensive, ulcerated lesion filling the antrum was visible through the operative incision in the mouth. Roentgenograms showed complete clouding of the maxillary sinus with extension of the opacity beyond the limits of the sinus, and destruction of the lateral antral wall and a portion of the malar bone. Roentgen therapy was instituted on September 13, 1939, with 220 kv., 3 to 4 mm. Cu filtration, 80 to 90 cm. distance. A total of 7,800 r (measured on the skin) was administered in ten days (20 sittings) through four portals as follows: (1) a left lateral portal, 64 cm.^{2} gradually diminished to 36 cm.^{2}—2,500 r; (2) a left posterior oblique portal, 48 cm.^{2}—1,450 r; (3) a left anterior portal, 52 cm.^{2} gradually diminished to 16 cm.^{2}—2,800 r; (4) a right oblique portal, 48 cm.^{2}—1,000 r. The daily doses varied between 550 and 1,000 r. The roentgen intensity varied from 2.8 r per minute to 6.2 r per minute. An "epithélite" appeared on the twelfth day and reached its height on the fifteenth day. The reaction disappeared on the twenty-eighth day. The height of the skin reaction was a pronounced erythema. Two months after the completion of treatment, extensive necrosis developed within the antrum and extended into the pterygoid fossa, resulting in complete trismus. The disease was uncontrolled, and one month later the patient showed signs of brain involvement and died December 24, 1939, four months after admission.

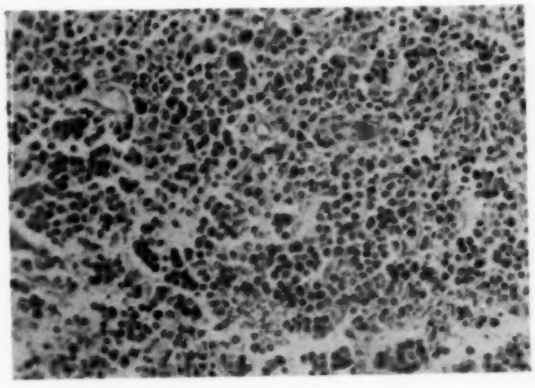

FIG. 6. Case III. Photomicrograph showing plasmocytoma.

Case III. M. S., female, white, aged fifty-six, admitted May 20, 1938. Pain in region of right antrum for five months, nasal obstruction in right side of nose for three months, frontal headaches and nasal bleeding for four months. Nasal polyps were removed in February, 1938, and were reported as being malignant. Examination disclosed a swelling over the right side of the face and nose. There was slight ptosis of the left upper eyelid. The right side of the nose was almost completely obstructed by a soft, ulcerated tumor. There was no adenopathy. Roentgen examination disclosed a soft tissue mass apparently arising in the right naso-antral wall. The upper portion of the medial antral wall showed signs of bone invasion. Clinically this lesion showed the features of a cancer of the antrum which had invaded the right nasal cavity and extended to the right half of the roof of the mouth with softening of the alveolar ridge but no ulceration of this structure. Microscopically there were masses of plasma cells, many of which were greatly enlarged. At other points the cells were round and polyhedral and varied in size and shape. There were numerous mitoses. The histopathologic structure led us to a diagnosis of plasmocytoma. The following roentgen treatment was given: 220 kv.; filtration, 3 mm. Cu plus 1 mm. Al; distance, 75 to 90 cm.; one right anterior field, 35 cm.^{2} gradually reduced to 12 cm.^{2} Daily dose varied between 200 and 750 r. Total dose—4,950 r (measured on the skin). Eleven treatments were given in ten days. Both eyes were protected during the treatment. An "epithélite" developed on the fifteenth day and disappeared on the thirty-first day. A moist "epidérmite" developed on the twenty-third day and healed on the thirty-fifth day. The tumor disappeared completely and there has been no evidence of disease three years and four months since treatment.

Case IV. F. S., female, white, aged fifty-one, admitted August 6, 1935. Pain in right side of head and face one year and five months. A Caldwell-Luc operation was performed and a specimen of tissue taken, which showed epidermoid carcinoma. There was a swelling of the right side of the face over the superior maxilla. The Caldwell-Luc incision was seen in the right upper alveolar ridge; the edges were necrotic and the antral cavity was filled with tumor tissue. The tumor invaded the naso-antral wall, and

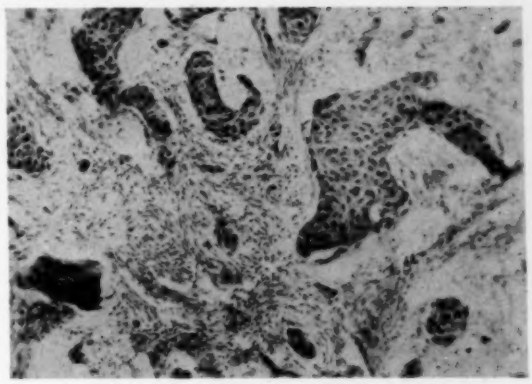

Fig. 7. Case IV. Photomicrograph showing epidermoid carcinoma.

presented in the right naris which it filled completely. Roentgenograms showed clouding of the right antrum, with destruction of the right naso-antral and inferior bony walls. The patient was treated by means of telecurietherapy (2 grams of radium) at 6 cm. distance through a 7 cm. round portal (one anterolateral field). She received 1,500 mg-hr. at each sitting, two sittings daily, giving 3,000 mg-hr. per day for ten days. Total dose—30,000 mg-hr. Treatment was begun on August 7, 1935, and completed August 16, 1935. The regression of the lesion was prompt and rapid. Six months later a small recurrent nodule appeared. This responded promptly to a small dose of radium. The patient remained free of disease for one year at which time she developed a small nodule on the right upper alveolar ridge which was treated by means of interstitial irradiation. Eight 1 mg. platinum needles were used (filtration 0.5 mm. platinum) for 120 hours. Total dose—960 mg-hr. The nodule disappeared and the patient has remained free of disease up to the present time, October, 1939.

DISCUSSION

The first case represents a lesion which has the clinical and microscopic features of radioresistance. It is probable that this tumor would not have responded to a less intensive course of treatment than that which was used. We have treated a number of carcinomas of the antrum similar to this one by the divided dose technique without success. The second case represents a fail-

ure due to the extent of the disease and the radioresistance of the lesion. The third case represents an excellent result which is explained by the radiosensitivity of the lesion. This result cannot properly be attributed to the new technique as it is probable that the lesion would have been eradicated by the older method. The radiosensitivity of the lesion in the fourth case is proved by the fact that a cure was obtained by a dose of radiation which was quite small and which, under general conditions, would be regarded as inadequate.

Of the four malignant tumors of the antrum, three were treated by a new technique of external irradiation which has been called the concentration method. The favorable result in one case is attributed to the new technique. The cases are presented to stimulate interest in the new method which is believed to offer important advantages in the control of the more radioresistant forms of cancer of the mouth, pharynx, larynx and accessory nasal sinuses. The report is also made with the view of calling special attention to the value of adequate external irradiation in the treatment of cancer of the antrum.

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CANCER OF THE CERVIX UTERI ASSOCIATED WITH PREGNANCY*

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THE coexistence of cancer of the cervix and pregnancy is sufficiently uncommon to warrant reporting its occurrence. The condition, when present, usually occurs in multipara, especially those in whom the pregnancies have occurred in rapid order. The diagnosis should offer no difficulty if proper regard is given to its possibility, and biopsy performed. Cases are easily missed, however, if all vaginal bleedings in early pregnancy are considered as threatened abortion and those in later pregnancy as placenta praevia, or other types of accidental hemorrhage, and, because of the conservative attitude toward these conditions, examination is postponed. Danforth reported 3 cases in 20,444 cases, Katz of Vienna 25 in 82,825 cases, Stoeckel 8 in 18,000 cases, Hirst 1 in 12,484 cases, and we are now reporting 4 in about 15,000 deliveries.

CASE REPORTS

CASE 1. G. L., white female, aged thirty-eight, gravida 9, para 8, last menstrual period, January 25, 1934. Admitted to the hospital on August 3, 1934, because of vaginal bleeding during the last five months of pregnancy. Examination revealed a well nourished woman in the eighth month of pregnancy. There was an irregular, ulcerated mass occupying the right half of the anterior lip of the cervix. Vaginal fornices and both parametria were free of disease (Stage 1, League of Nations Classification). Biopsy of cervical mass revealed "epidermoid carcinoma (middle maturity)" (Fig. 1). Operation was advised but was refused by the patient, who left the hospital. She returned on August 15, 1934, twelve days later, at which time the neoplasm of the cervix was found to be about double its previous size. Dr. Aranow, Director of the Obstetrical Division, performed a Porro-cesarean operation on August 21, 1934. As much of the carcinoma of the cervix as possible was removed by cautery and the cervical lips were then sutured together. The vagina was packed with 3 per cent iodine gauze. The abdomen was then opened and a cesarean section, total hysterectomy, and bilateral salpingo-oophorectomy were done. A seven month fetus was extracted which survived only six days. The postoperative course was uneventful. Roentgen therapy (300 kv., 4 ma., 2 mm. Cu filter, 80 cm. target-skin distance) was administered from September 4 to November 1, 1934, during which time a dose of 2,000 r† was given to each of three pelvic portals measuring 12 by

FIG. 1. Photomicrograph of tissue removed at biopsy in Case 1 (×880). "Epidermoid carcinoma (middle maturity)." Cells are large and irregular in size.

† International roentgen unit measured in air.

* From Tumor and Radiation Therapy Service and Obstetrical Division of Morrisania City Hospital, New York City.

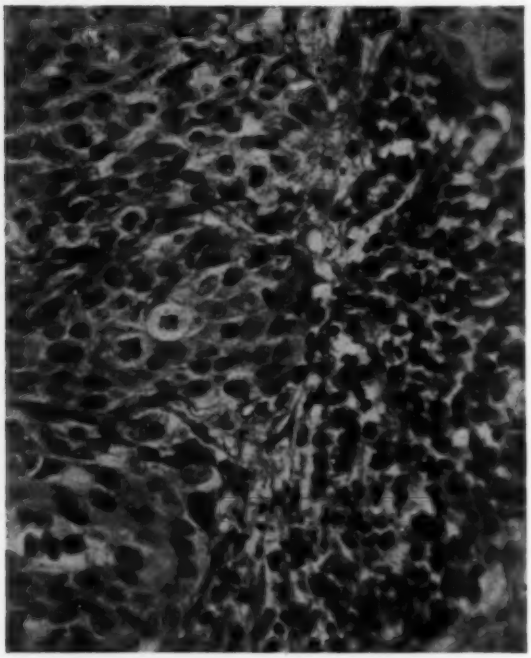

FIG. 2. Histopathological appearance of tissue removed in Case II (×880). "Epidermoid carcinoma (middle maturity)." Several mitotic figures are seen.

15 cm. Patient has now been well for over six years.*

CASE II. C. G., Female, Negress, aged forty, gravida 9, para 8, last menstrual period, July 2, 1934. Admitted to the hospital because of labor pains, and premature child of seven months was delivered on February 5, 1935. No previous abnormal symptoms or vaginal spotting. Blood Wassermann reaction was negative. The child died shortly after birth. There was no unusual bleeding during delivery and puerperium was normal. On routine postpartum examination on February 14, there was noted a hard, mushroom-like new growth situated on the anterior lip of the cervix, which almost filled the transverse diameter of the vagina. The neoplasm extended onto the anterior vaginal wall. Parametria normal (Stage II). Biopsy was taken and reported as "epidermoid carcinoma, middle maturity" (Fig. 2). Roentgen therapy (300 kv., 4 ma., 2 mm. Cu filter, 80 cm. target-skin distance) was given from February 18 to April 5, 1935, the dosage being 2,300 r to each of four pelvic fields, 8 by 15 to 12 by 15 cm. in size, directed towards the cervix. The lesion in the cervix received approximately 5,000 r. On April 6, intra-uterine radium (active length of applicator 6.0 cm.) was inserted for a dose of 3,100 mg-hr. in sixty-nine hours. From April 10 to 17, vaginal radium, with two corks, was given in two sessions for a total dose of 4,200 mg-hr. The cervical mass diminished in size by 75 per cent after one-half of the total roentgen therapy had been delivered. All gross evidence of disease disappeared by June 26, 1935. On August 19, 1935, patient complained of dysuria but this symtom was no longer present one month later. The patient was lost to observation until July 15, 1940, five years after treatment, at which time she entered another hospital with extensive carcinoma involving the entire pelvis.

CASE III. F. P., white female, aged thirty-two, gravida 10, para 4; five induced abortions; last menstrual period August 20, 1935. Admitted to the hospital on November 20, 1935, complaining of vaginal bleeding after coitus and foul vaginal discharge, all of one month duration. Examination revealed a well nourished

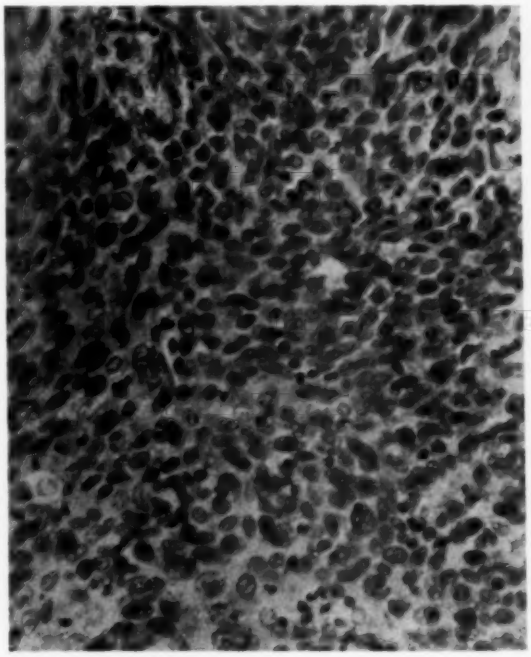

FIG. 3. Histopathological appearance of tissue taken at biopsy in Case III (×880). "Transitional type of squamous cell carcinoma." Cells are irregular in size. Numerous mitotic figures present.

* The patient was seen in July, 1942, and at that time was still free of disease.

woman, three months pregnant. The entire cervix was transformed into a large cauliflower-like mass which bled easily. Lower uterine segment was freely movable, and there was no parametrial involvement (Stage I). Biopsy of the tumor was reported as "transitional type of squamous cell carcinoma" (Fig. 3). Roentgen therapy (180 kv., 5 ma., 0.5 mm. Cu filter, 50 cm. target-skin distance) was given from November 23 to December 20, 1935, the dosage being 1,100 r to each of three anterior and three posterior fields, 10 by 15 cm. in size, directed towards the cervix. The lesion in the cervix thus received approximately 2,500 r. Vaginal radium was used from November 29 to December 17, 1935. Thirty milligrams of radium, distributed in two corks, were inserted for periods varying from six to twenty-three hours until a dose of 1,820 mg-hr. had been given. These treatments were carried out on the same days as the roentgen therapy, the radium being removed just before the first, and replaced shortly after the last roentgen treatment of the day. On December 18, there was almost complete disappearance of the new growth on the cervix except for an area 2.0 cm. in diameter. The patient had complained of extreme discomfort during the insertion of the radium into the vagina and, at this point, refused to allow a continuation of this treatment. Accordingly, on December 20, the residuum in the cervix was implanted with seven radon gold seeds, 1.2 mc. each, distributed over an area 3.0 cm. in diameter, the dosage being 6,200 r_γ. On December 30, patient aborted, spontaneously, a four month fetus. Intra-uterine radium was used on January 24, 1936, the dose given being 3,000 mg-hr. On February 3, patient was free of all symptoms, and there was no gross evidence of disease. Three months later, however, there was marked peri-urethral thickening, and an ulceration of the cervix. On July 28, 1936, patient was re-admitted to the hospital because of severe pain in back and in both lower extremities. The entire pelvis was filled with carcinomatous growth. Patient died in another institution on August 28, 1936, nine months after the first treatment. There was no autopsy.

Case IV. C. B., white female, aged thirty-eight, gravida 9, para 8, last menstrual period May 28, 1932. Admitted to the hospital on February 9, 1933, because of pregnancy and labor. Examination revealed a very obese woman in the eighth month of pregnancy. Blood

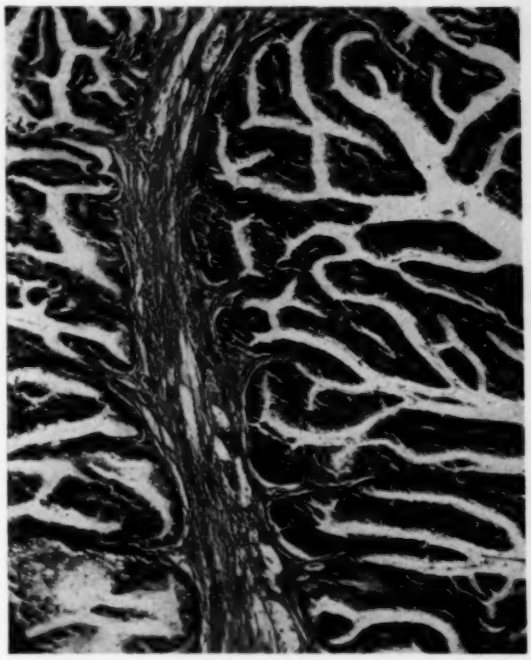

Fig. 4. Photomicrograph of tissue removed at biopsy in Case IV. (×200). "Malignant adenoma." Acini are regular. No invasion of basement membrane.

Wassermann reaction was negative. A stillborn child was delivered on the day following admission. There was no excessive hemorrhage. During parturition, a friable, bleeding mass was noted on posterior lip of cervix. Biopsy of this mass was reported as "malignant adenoma"* (Fig. 4). Patient refused treatment and left the hospital. Since the vaginal bleeding persisted, she returned to the hospital on March 13, 1933. Examination at this time revealed the posterior lip of the cervix to be converted into a large cauliflower-like mass extending into the posterior vaginal fornix and involving the upper one-third of the posterior vaginal wall. Parametria were normal (Stage II). Another biopsy taken at this time showed the same histopathological structure as on the previous occasion. Because of the extreme obesity of the patient, approximately 40.0 cm. in anteroposterior diameter of the lower abdomen, roentgen

* Regarding adenoma of the cervix, Ewing says that while many of these tumors are benign, some recur locally and have produced metastases in the vagina. He adds that Winter interprets these tumors as a slowly developing adenoma of cervical glands with carcinomatous tendencies. Stone stated: "It seems not improbable that such a process [adenoma] is a prototype of that infrequent form of adenoma of cervix, in which the mucus cells preserve their normal type after the glandular growth has reached bulky proportions and become clinically malignant."

therapy was considered to be ineffective. Radium therapy was therefore instituted on March 21. The uterine cavity was treated with a tandem containing 30 mg. of radium filtered with 1.0 mm. Pt (active length 6.0 cm.), for seventy-eight hours, for a total of 2,340 mg-hr. At the same time, a similar amount of radium, filtered with 2.0 mm. Pt, distributed in three vaginal corks, was placed in vagina for a dose of 2,340 mg-hr. On March 25, four vaginal corks, containing a total of 60 mg. of radium, were left in situ for a dose of 4,470 mg-hr., making a total of 6,810 mg-hr. in the vagina. Patient was symptom-free until February 8, 1935, when vaginal bleeding recurred. Examination failed to reveal the cause of this bleeding. Dilatation and curettage of the uterus was done on March 13, 1935. The removed tissue showed "glandular hyperplasia." Radium was inserted into the uterus for a dose of 2,500 mg-hr. The patient has been perfectly well to date, May, 1941, a period of over eight years since treatment for carcinoma of the cervix.*

DISCUSSION

We are especially interested in the following questions which have been the subject of much discussion.

What is the effect of the carcinoma of the cervix on pregnancy and how does pregnancy affect the cancer? Is delivery possible *per vias naturales* without extreme risk to the mother from hemorrhage or infection? Should patients be operated on and what should the nature of the operation be? Should irradiation be given with fetus *in utero?*

In one patient, seven months pregnant, the tumor of the cervix was found to increase to about twice its size within a period of twelve days. Another patient, three months pregnant, with Stage I disease, treated with irradiation, died about eight months after abortion had occurred. We believe that pregnancy may hasten the progress of the carcinoma. We are not in accord with the opinions of those who believe that the termination of pregnancy may release an inhibiting factor to the growth of the carcinoma.[2,16] In fact, with the one exception quoted above, our experience has been very much to the contrary. One patient, with Stage I disease, who had a Porro-cesarean in the eighth month of pregnancy, followed by roentgen therapy, has been free of disease for over six years. Of two other patients, both treated shortly postpartum, one (Stage III) survived five years and was dying of carcinoma, while the other (Stage II) has been free of disease for over eight years.

DeLee states: "Since labor creates a frightful (45%), immediate mortality from rupture of the uterus, hemorrhage, sepsis, exhaustion and shock (when carcinoma of the cervix is present), labor pains are not to be awaited but a Porro-Caesarean done." Yet, in 2 of our cases, in the eighth month of pregnancy, delivery *per vaginam* occurred with no complications whatever. In each case only one of the lips of the cervix was involved, which may account for the relative ease with which the patients gave birth. Other authors have reported similar experiences.[11,17] This does not mean to say that one should allow delivery *per vaginam* to occur as the method of choice but rather to emphasize that such delivery may occur spontaneously, and sometimes without danger. We believe that only by studying numerous individual case reports can one arrive at a conclusion. In spite of the good result obtained with a Porro-cesarean plus roentgen therapy in Case II, study of the literature fails to reveal any improvement in end-results if a Porro operation is done rather than the classical cesarean section. We therefore believe that a simple cesarean section plus adequate radiation therapy should be the treatment of choice.

Radiation Therapy. The stage of pregnancy plays a decided rôle in the choice of treatment of the carcinoma.

From a study of the literature and from personal experience, we have reached the following conclusions as to the preferable technique:

* The patient was seen in July, 1942, and was still free of disease.

A. During First Trimester.

1. *Radium in the vagina.* Radium should be applied in as large amounts as possible, and for only eight to twelve hours continuously for any one session. This technique is suggested so that the total dose may be given in as few applications as possible and so that the packing of the vagina for only a small number of hours will minimize the tendency to abortion. Frequent insertions of the radium have not provoked an abortion in our case, and since the procedure is always done under sterile precautions, the danger of infection is minimized. The action of the radium on the infected, bleeding carcinoma will usually cause it to heal over more or less completely at the end of about four to six weeks. Since the abortion resulting from the roentgen therapy (see below) does not occur until the above period of time has elapsed, the danger of sepsis and hemorrhage during the abortion is markedly minimized. This is the chief reason for giving the radium therapy as soon as possible after the diagnosis has been made.

Technique: Corks, measuring 3.2 cm. in longest and 1.8 cm. in shortest diameter, are inserted into the vagina against the new growth on the cervix. The number of corks employed is usually two, although one or three may be used depending upon the size of the vagina and the extent of the disease. Radium filtered with 2.0 mm. Pt is contained in each cork. The rectum and urinary bladder are displaced away from the corks by gauze packing. The total dose given varies from 3,000 to 6,000 mg-hr., depending upon the number and thickness of corks used, the extent of the disease, as well as upon the amount of roentgen radiation one calculates to give to the cervix.

2. *Roentgen therapy.* This should be given alternately with the radium therapy. The roentgen treatments may be given in the morning and afternoon. The radium is then inserted into the vagina and left in place until the next morning. It is removed before the roentgen therapy is given again, reinserted upon the completion of this therapy in the afternoon, and so on until the desired radium dosage has been delivered. Spontaneous complete abortion will usually occur within four to six weeks after the beginning of the roentgen therapy. Surgical abortion is rarely necessary or indicated.

Technique: The physical factors employed are 180–200 kv., 5–20 ma., 0.5 to 2.0 mm. Cu filter (0.9 to 1.8 mm. Cu half-value layer), 50 to 80 cm. target-skin distance, and portals varying in size from 6 by 15 to 10 by 15 cm. The fields used are anterior and posterior pelvic directly over the uterus, and two additional ones on each side of the central portal, anteriorly and posteriorly. The latter four portals are approached by a ray which is angled mesially 30 degrees, so that the main portion of the enlarged uterus will not be missed. At the same time, the parametria are irradiated by the treatment of those fields. Treatments are given twice daily, with doses varying from 100 to 200 r at each seance. The exact dose that each portal receives will vary with the size of the patient and of the field, extent of the disease, and general condition of the patient. As a rule, however, the dose per portal totals from 1,000 to 2,000 r. If extensive parametrial infiltration is present, the number of portals on the involved side or sides of the pelvis is increased by the addition of fields over the greater sciatic notch, the inguinal ligament and the lateral pelvis.

3. *Radium in the uterus.* Several weeks after the abortion from the roentgen therapy has occurred, the third and final stage of the treatment is carried out. At this time, a rubber tube containing that amount of radium which is available for the purpose (usually 50 to 100 mg., filtered with 1.0 mm. Pt) is inserted into the uterus. The usual dosage is about 4,000 mg-hr., although this may vary according to the size and length of the uterine cavity.

4. *Additional or supplementary therapy.* Occasionally, there may be residual carcinoma in the cervix after the above described procedures have been carried out.

There then remains the choice of using implantation of needles or gold radon seeds directly into the area of disease, or of the employment of intravaginal roentgen therapy. If the implantation is done, needles or seeds, the dose is calculated in gamma-ray roentgens as described by Paterson and Parker. The total dose varies from 4,000 to 6,000 r_{γ}, depending upon the extent of the disease and previous treatment. With shockproof roentgen therapy machines now available, there exists a very simple and quick method of inserting a cone into the vagina and focusing the roentgen-ray beam directly upon the involved area in the cervix. The dosage to be given by this method depends upon the size of the portal, and the amount of radiation already administered. Usually, however, a field of 2 to 3 cm. in diameter should be given about 6,000 r. We use the same physical factors as previously described, except that the distance is 35 cm. The size of the portal necessarily depends upon the caliber of the cone that can be introduced into the vagina.

TABLE I

OUTLINE OF TREATMENT IN CARCINOMA OF CERVIX UTERI ASSOCIATED WITH PREGNANCY

First Trimester	*Second Trimester*		*Third Trimester*
	First Half	*Second Half*	
(1) Vaginal radium alternately with (2) Roentgen therapy (3) Intrauterine radium	(1) As in first trimester except for (2) Surgical abortion if spontaneous abortion does not occur	(1) Vaginal radium (2) Cesarean section as soon as possible (3) Roentgen therapy (4) Intrauterine radium	(1) Cesarean section as soon as possible, followed by roentgen, vaginal and intrauterine radium therapy (2) Pending operation, vaginal radium

B. During Second Trimester.

The treatment followed during this period depends upon whether or not the uterus has already reached the size of four and one-half months gravidity.

If the size of the uterus is less than this, treatment is carried out as in the first trimester. However, since the more advanced stage of pregnancy makes the abortion from roentgen therapy at this time less certain, a surgical abortion is advised if the uterus has not emptied itself spontaneously within four to six weeks after treatment has been started. This is especially indicated if the uterus continues to increase in size two to three weeks after the beginning of roentgen therapy.

However, if the uterus is past the stage of four and one-half months gravidity, roentgen therapy is omitted at first. First, because of the definite danger to the fetus, should it survive, and second, because it may be impossible, with this procedure, to produce a roentgen abortion. The suggested treatment then is as follows:

Vaginal radium as in first trimester. In the latter months of pregnancy, the fetus is usually at such a great distance from the cervix, and thus from the radium in the vagina, that the possibility of producing radiation damage to it is quite remote.

Should spontaneous delivery occur within a few weeks, roentgen therapy is given as soon thereafter as the condition of the patient allows. Intra-uterine radium therapy, as in the first trimester, is administered as soon as possible after completion of the roentgen therapy.

If spontaneous delivery does not occur, one then awaits the moment when the life of the child may be assured, when a simple cesarean section should be performed. Subsequent treatment is as above (roentgen therapy followed by intra-uterine radium).

Technique of roentgen therapy: This is essentially the same as described previously, with the following exceptions:

(1) Two anterior and two posterior pelvic portals are used, usually 10 by 15 cm. in size. The skin is protected for a distance of 2.0 cm. on each side of the midline. As stated previously (A-2), additional fields are used if extensive parametrial involvement is present.

(2) The rays are directed at right angles to the portal.

(3) The total dose to each field varies from 1,500 to 2,500 r.

C. During the Third Trimester.

Cesarean section should be done at the most opportune moment compatible with the life of the baby. If the patient is seen prior to this time, one should apply vaginal radium as previously described. Then one waits until the cesarean section can be performed. Following the operation, treatment is carried out with roentgen radiation and intra-uterine radium as in the latter part of the second trimester.

Should the cesarean section be done first, subsequent treatment is as follows:

(1) Roentgen therapy as soon as the patient has recovered from the operation (technique is similar to that described in treatment during latter half of second trimester).

(2) Vaginal radium. The technique is similar to that previously described (A-1). Here, however, only 10 to 15 mg. of radium are inserted into each cork so that the treatment may be prolonged over five to seven days. The corks are removed every forty-eight hours. The total dose is approximately 4,000 to 6,000 mg-hr.

(3) Intra-uterine radium (the technique is as described in A-3).

D. During Postpartum.

Treatment is carried out according to routine methods. Our usual procedure is to give roentgen therapy to the pelvis, followed by intra-uterine and vaginal radium treatment (see under third trimester).

SUMMARY

Four cases of carcinoma of the cervix uteri associated with pregnancy are presented. One of these cases was an adenoma which was clinically malignant.

In one of these patients there was a visible increase in growth of the carcinoma during pregnancy.

Two patients were delivered *per vaginam* without complications.

All patients received radiation therapy.

Three of our patients have survived over five years, one with recurrent carcinoma.

Based upon study of the literature and on personal experience, recommendations for the treatment of carcinoma of the cervix uteri associated with pregnancy are suggested.

We wish to thank Dr. Harry Aranow, Director of Obstetrics and Dr. William Harris, Chief of Tumor and Radiation Therapy Service, for their kind aid and cooperation.

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A NEW DESIGN FOR INCREASING THE HEAT-DISSIPATING CAPACITY OF ROTATING ANODE TUBES

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THE problem of heat dissipation from the anode system of a rotating anode tube differs from that involving the conventional stationary anode tube in that heat transfer to the exterior of the tube by conduction through the anode structure is almost entirely precluded by the bearings upon which the rotating anode is supported. This problem has received the attention of roentgen tube designers for many years, and there are numerous suggestions in the technical[1,2] and patent[3,4,5,6] literature of means to obtain rapid heat dissipation from the rotating anode. Certain current trends in diagnostic practice, namely, so-called "spot film" or "serialograph" work, and photoroentgenography for rapid chest surveys, both of which call for greatly increased roentgen-ray energy requirements, have recently focused still greater emphasis on this factor in tube design.

All commercially successful designs of rotating anode tubes have relied on the principle that heat dissipation must take place by radiation from the surface of the anode structure. In practice, the construction of rotating anodes consists of a copper portion, adapted to act as the rotor of an induction motor, which is supported on the bearings and in turn supports at its extreme end the tungsten target itself. There are two principal variations in design: (1) in which the target is in intimate contact with the copper rotor portion; (2) in which the target is supported on a slender stem of refractory metal so as to provide a path of low heat conductivity between the target and the copper rotor.

In anode structures of the first-mentioned variety, the high degree of heat conductivity between the respective parts results in the entire structure assuming a practically uniform temperature and the total surface becomes the medium of heat dissipation by radiation. The limit to the maximum amount of radiation obtainable is determined by the maximum permissible operating temperature of the copper, the coefficient of thermal emissivity of the surface, and the dimensional limitations of the surface area from a practical point of view. A number of expedients have been suggested to increase the radiation factor for the entire surface of the anode structure.[5,6] The customary practice is to make the surface area as large as is consistent with convenient over-all dimensions of the entire tube, and to artificially blacken the copper surfaces to increase the radiation factor.

In the second type of construction, by far the greater part of the radiation takes place from the target member of the anode assembly and very little from the copper portion. In considering heat dissipation from this type of anode, it has generally been assumed that the conduction of heat from the target into the copper rotor is negligible. Various expedients have been suggested for reducing the heat conductivity of the connecting member to its lowest possible limit, and the heat-dissipation ratings of the tubes designed along these lines have been based on the radiation characteristics of the tungsten target alone.[7,8] Thus, theoretically, since the all-tungsten target can be raised to a very high temperature without damage to itself, an extremely high rate of heat dissipation by radiation can be attained therefrom.

However, when conditions of practically

continuous loading, as in roentgenoscopy, are encountered, it has been found in prior tubes of this construction that the continuous heat-dissipation rating is limited not by the radiating capacity of the tungsten member but by the maximum allowable temperature of the copper portion of the structure. Unavoidable conduction of heat through the supporting member, when the

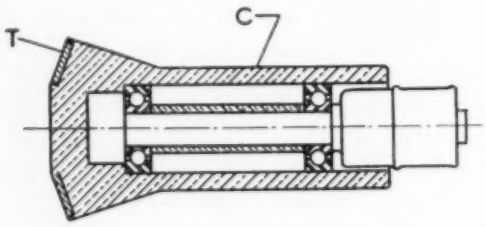

FIG. 1. Schematic diagram of rotating anode structure in which target is in intimate contact with copper rotor.

target is at the high temperature for rapid radiation, eventually results in excessive temperature of the copper rotor. Thus the rating for this type of operation which can be assigned to such tubes, while greater than that for tubes of the first-mentioned variety, is still less than is desired for some applications.

In the development of a design to overcome this limitation, instead of attempting to keep the copper rotor from exceeding permissible temperature limits by reducing the conductivity of the connecting member as much as possible, attention was directed to improving the radiation factor of the copper surface while restricting the conductivity of the connection within certain limits. Under these conditions, it was found that the increase in radiation from the copper rotor makes possible a much greater gain in the radiation from the target. The extent of this gain is revealed by means of a mathematical analysis of the thermodynamics involved, as discussed in the succeeding paragraphs. This analysis also involves a comparison of this type of construction with that in which the tungsten target and the copper rotor are in intimate contact.

Figure 1 illustrates schematically the type (1) anode system and Figure 2 illustrates type (2). Both types may be considered as consisting of a tungsten member "T" and a copper member "C", joined together by a thermal path of conductivity K. The mathematical expression for the total heat energy radiated from such a structure is

$$E = R_t(T_t^4 - T_o^4) + R_c(T_c^4 - T_o^4)$$

where E = energy radiated
R_t = radiation factor for tungsten member (dependent on area and coefficient of emissivity)
R_c = radiation factor for copper member
T_t = absolute temperature of tungsten member
T_c = absolute temperature of copper member
T_o = absolute temperature of surroundings.

If, as in the practical cases being considered, T_o is less than one-half the value of T_t and T_c, the error involved in ignoring T_o is less than 6 per cent, and it is permissible to eliminate T_o and thus simplify the expression, as follows:

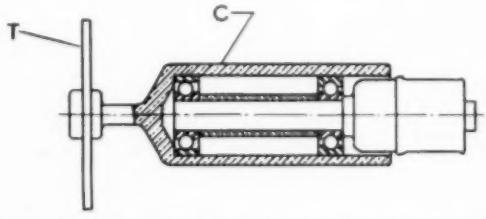

FIG. 2. Schematic diagram of rotating anode structure in which target is separated from copper rotor by a member having low thermal conductivity.

$$E = R_t T_t^4 + R_c T_c^4$$

The input energy is all applied to the tungsten member. Hence, in equilibrium, the energy radiated from the copper is equal to that conducted into it from the tungsten through the thermal path connecting them. Therefore, $R_c T_c^4 = K(T_t - T_c)$, K being the conductivity of the connecting

path. From this expression $T_t=(R_c/K)T_c^4+T_c$, which permits us to express E as a function of T_c as follows:

$$E=R_t((R_c/K)T_c^4+T_c)^4+R_cT_c^4$$

To illustrate graphically how E varies with T_c under various conditions, several curves based on the above equation are plotted, all of which relate to an anode structure having a tungsten member with a surface area of 80 sq. cm. and a copper member with a surface area of 100 sq. cm., which figures represent a structure of practical dimensions. The radiation factor for a smooth tungsten surface of 80 sq. cm. is determined from published data, giving $R_t=1.17\times10^{-10}$. The radiation factor for copper depends on the condition of the surface. This factor has been determined experimentally for a surface of 100 sq. cm., both in a polished condition and in a blackened condition for maximum radiation. The values are

$R_c=6.85\times10^{-11}$, polished
$R_c=2.95\times10^{-10}$, blackened

The factor K is also subject to variation, depending on the manner in which the two members are joined together. If the tungsten makes the best possible thermal contact with the copper, the two elements are at practically the same temperature, and K can be considered equal to infinity. If the members are joined through a third member whose conductivity is purposely reduced to a minimum, a practical value of K has been found to be .044.

In Figure 3, the factor K is taken as infinity, representing the construction of type (1), and E (total radiated energy) is plotted as a function of T_c (temperature of the copper member), both for the condition where the copper is polished ($R_c=6.85\times10^{-11}$), and for blackened copper ($R_c=2.95\times10^{-10}$). In Figure 4, K is taken as .044, representing the construction of type (2), and E is plotted as a function of T_c for both polished and blackened copper. A value of 700° K (427°C) may be taken as the upper limit of a safe value for T_c. A comparison of Figures 3 and 4 will indicate the disproportionately great gain resulting from blackening the copper rotor in the construction of type (2) as compared with type (1). In making the comparison, it should be borne in mind that in actual practice the area of the blackened copper surface employed in the construction of

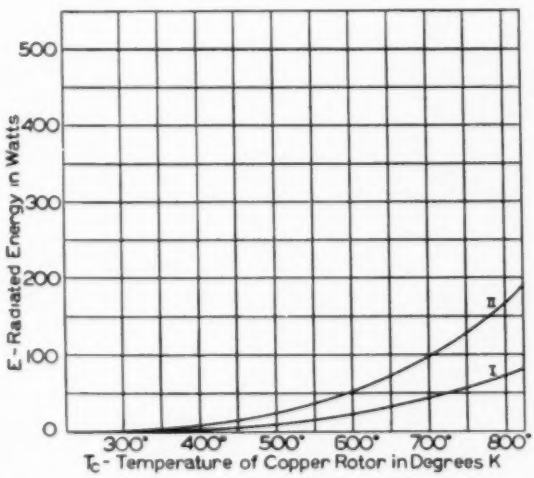

Fig. 3. Radiated energy as a function of temperature of copper rotor when target is in intimate contact with copper. Curve I for polished copper. Curve II for blackened copper.

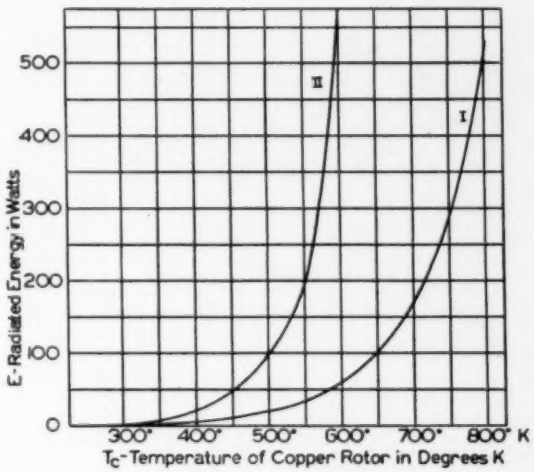

Fig. 4. Radiated energy as a function of temperature of copper rotor when target is separated from copper by a path of thermal conductivity $K=.044$. Curve I for polished copper. Curve II for blackened copper.

type (1) is usually somewhat greater than that assumed in this example and hence the total dissipation obtainable from such a tube will be somewhat greater than the value of 95 watts indicated at $T_c = 700°$. However, it is interesting to note (not indicated in the charts) that if the radiating area represented in Figure 3 should be increased to obtain the value of energy radiated under the conditions represented by Figure 4, copper blackened, at $T_c = 700°$, an area of approximately 2,200 sq. cm. would be required. This figure is to be compared with a total area of 180 sq. cm. assumed in this example.

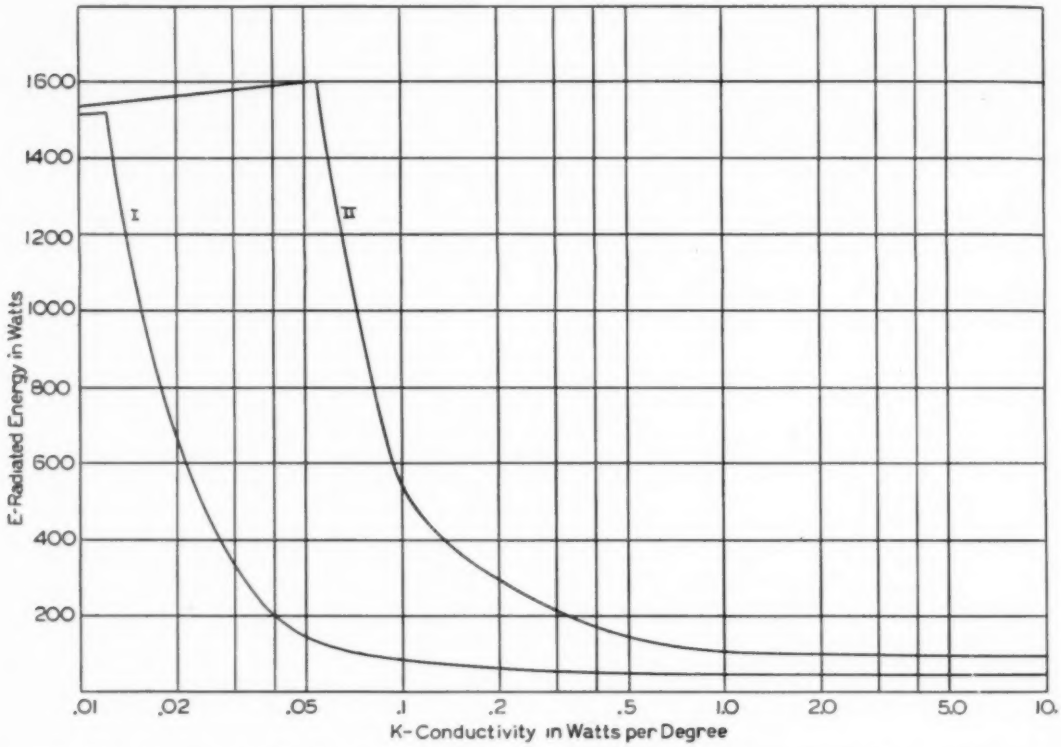

FIG. 5. Radiated energy as a function of conductivity between target and copper rotor, for a maximum target temperature of 1,900° K and maximum copper temperature of 700° K. Curve I for polished copper. Curve II for blackened copper.

To determine the range in which the effect of blackening the rotor is a disproportionate increase in heat-dissipating capacity, curves are also plotted with E as a function of K (Fig. 5). In the equation $E = R_t((R_c/K)T_c^4 + T_c)^4 + R_c T_c^4$, T_c is taken as 700°, R_t and R_c are taken as the values previously assumed, and E is computed for various values of K thoughout the range indicated in Figure 5. For those values of K which would result in T_t (which equals $(R_c/K)T_c^4 + T_c$) exceeding 1,900° absolute, it is assumed that T_t must be limited to this value and the value of E is computed on this basis rather than on the basis $T_c = 700°$. Thus, the two curves of Figure 5 represent the radiation obtainable for various values of K when a maximum limit is placed on T_t of 1,900° and T_c of 700°, both of which may be considered as practical maximum working values. Curve I is for a polished copper rotor and curve II for a blackened copper rotor.

It will be noted that, for a structure of these particular dimensions, the range of values for K in which blackening the rotor results in a disproportionately high increase in total radiation lies between approximately .012 and .5, K being expressed in

watts of energy conducted per degree of temperature difference between the tungsten and copper members. The most advantageous condition occurs when K is approximately .05, which value can readily be secured in practice. Values lower than .04 would be somewhat difficult to obtain in practice, without exceeding desired dimensional limitations or sacrificing desirable characteristics for the conducting member.

If dimensions different from those assumed in the above example are chosen for the two principal elements of the rotating anode structure, curves relating to such a structure plotted in the manner of Figure 5 will be generally similar in form and shape to those of Figure 5, but the critical values of K will be different from the values .012 and .5 which were ascertained in this example. Further analysis indicates that the factor R_c/K in the equation for E is the factor whose value will determine the significant points of the curves. R_c is directly proportional to the area of the surface of the copper rotor, and hence the limits for the value of K resulting in disproportionate increase in total radiation produced by blackening the rotor are directly proportional to the area of the rotor. Therefore the limiting range for K is .00012 A to

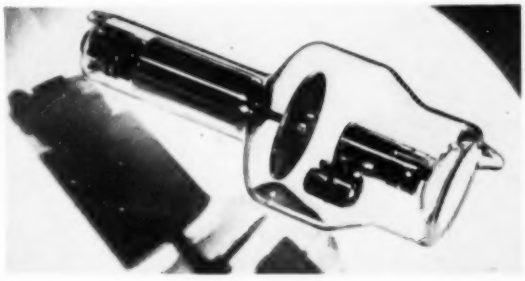

Fig. 6. Rotating anode tube constructed according to the principles enunciated in this article.

.005 A, A being the area in square centimeters of the surface of the copper member of the anode assembly. The most advantageous value for K is approximately .0005 A.

The above discussions pertain to the rotating anode roentgen tube only, without reference to any rayproof or shockproof enclosure in which the tube may be mounted. Further limitations may be imposed by the provisions for cooling the tube incorporated in such enclosures, and the temperature limitations of oil or other insulating materials and component parts involved in the enclosure. As a practical matter, the maximum rating of tubes incorporating this improvement is limited by the heat-dissipating capacity of the shockproof assembly rather than that of the tube itself.

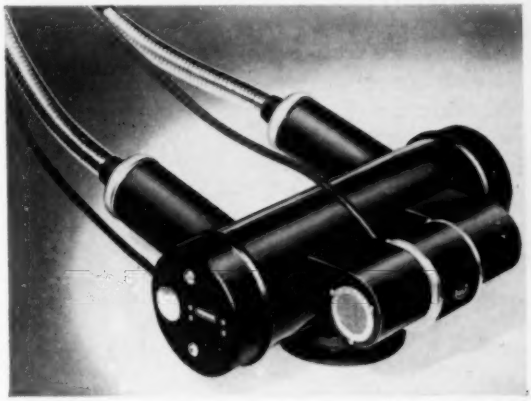

Fig. 7. Shockproof rotating anode tube unit designed for maximum over-all heat dissipation.

Practical working ratings for roentgenoscopy have now been assigned to a shockproof oil-immersed rotating anode tube constructed according to this principle of 5 ma. at 85 kv. (peak) for thirty minutes continuously, or indefinitely for intermittent operation in which the rest periods are equal to the operating periods, of ten minutes maximum. No artificial cooling means is necessary to obtain these ratings, which are equal to those of the best stationary anode shockproof tubes in this respect.

Addendum. Since the original date of submission of this article, a further development in shockproof enclosures for this type of tube has made possible the more nearly complete utiliza-

tion of the heat-dissipating capacity of the tube itself. The shockproof unit now available is equipped with an air-circulator to increase heat dissipation from the surface of the enclosure. In this form the tube has a rating for roentgenoscopy of 5 ma. at 85 kv. (peak) continuously without time limitation, and corresponding capacity for rapidly repeated roentgenographic exposures continued for long periods of time. For use in mass photoroentgenography, for example, operation involving high-energy exposures (200 ma. at 90 kv. for 0.2 second) can be carried out with this tube at a rate of as many as six exposures per minute, continued indefinitely.

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EDITORIALS

METAL FUME FEVER AND NITRIC FUME PNEUMONIA

With the great increase in the amount of arc welding being done at the present time it may be opportune to review present concepts of the two clinical entities commonly known as metal fume fever and nitric fume poisoning—both sometimes erroneously spoken of as "welder's pneumonia."

Metal fume fever appears to be a clear-cut clinical entity consisting chiefly of a one day febrile reaction, with cough, developing after exposure to zinc or magnesium fumes, incidental to arc welding. It usually occurs on the first or second day of work, after the welder has been laid off or absent from work for a short time. Johnstone[1] observes: "Many workers report a state of well-being during the week, but after a week-end they will notice various grades of reaction upon a return to work." Experienced welders appear to pay little or no attention to it, taking it for granted as one of their occupational inconveniences. It is most apt to develop in those working in poorly ventilated or close quarters, welding or burning materials composed largely or in part of zinc or magnesium, such as galvanized pipe. It is important to note that the condition is of very short duration and subsides spontaneously. There are *no* demonstrable roentgen changes in the lungs.

Nitric fume poisoning and nitric fume pneumonia are very different and potentially much more serious disorders. They occur in welders and other metal workers exposed to large amounts of nitric oxide and nitrogen peroxide fumes. For the term "nitric fume poisoning" we are indebted to Beard.[2] Nitric oxide is said to be thirty times as poisonous as carbon monoxide. On contact with human tissues it becomes largely nitric acid. The immediate effects of exposure to large amounts of nitric oxide or nitrogen peroxide are pulmonary congestion and edema. These patients are usually too sick for roentgen examination, but if examined would probably show the ordinary changes of pulmonary edema such as one sees in certain types of nephritis and following coronary occlusion. If no complicating bronchopneumonia or lobar pneumonia develops, and the exposure has not been extraordinarily severe, the changes will subside completely, and there will be no late roentgen findings. If, however, there is a complicating bronchopneumonia or lobar pneumonia, then there may of course be some post-pneumonic changes such as small areas of unresolved inflammation, scars or localized pleural thickening.

As far as can be ascertained, there are no roentgenologically demonstrable late changes following exposure to metal vapors, despite reports of diffuse pulmonary nodulation by one or two observers. The illustrations of the roentgenograms in these reported cases showed no convincing evidence of nodulation, nor changes acceptable as "pulmonary fibrosis" to experienced roentgenologists. The clinical manifestations of nitric oxide or nitrogen peroxide pulmonary edema or inflammation are largely those of dyspnea and cyanosis. If secondary infection supervenes, the findings change accordingly to those of a more or less severe pneumonia.

We have recently observed five cases of afebrile pulmonary consolidation in persons exposed to welding fumes, but the relationship of the lobular densities to the apparent

[1] Johnstone, Rutherford T. Occupational Diseases. W. B. Saunders Co., Philadelphia, 1941.

[2] Beard, R. B. Personal communication.

fume exposure is far from established. They may well be due to patches of unresolved pneumonia following ordinary coccal or virus infections.

It is important that the term "welder's pneumonia" be avoided since there is no specific pneumonia of welders as such, and the term gives rise to a misconception both in medical and lay minds. The correct terms would be "zinc fume fever" or "magnesium fume fever" for the transient illness, and "pneumonia complicating nitric fume poisoning" for the more serious entity.

L. Henry Garland

Underwood & Underwood

HOMER CLYDE SNOOK
1878–1942

THE death of Dr. Clyde Snook has caused further lamentable but inevitable attenuation of that renowned group of scientists who, through their epochal accomplishments of an earlier day, contributed so bountifully to the steady and sound development of American radiological practice. Concerning, for example, the influence of both Snook and Coolidge on this amazing expansion with particular reference, respectively, to the rotary "interrupterless" x-ray transformer and the hot-cathode x-ray tube, it has been accurately stated* that "these two discoveries

* Dr. George M. MacKee in "The Science of Radiology," Charles C Thomas, Springfield, Ill., 1933.

revolutionized roentgenology." Of the truth of this assertion there can be no doubt.

Clyde Snook, internationally known electrophysicist and engineer, for the past forty years a highly esteemed active member of the American Roentgen Ray Society, was born in 1878 at Antwerp, Ohio, a son of Wilson Hunt and Nancy Jane (Graves) Snook. In 1903 he married Miss May Eusebia McKee, of Warren, Ohio, who survives him. His graduation in Arts (1900) from Ohio Wesleyan University was followed by a period of teaching physics and chemistry at Xenia, Ohio, and also at Allegheny College in Meadville, Pennsylvania, where he became Assistant Professor of Chemistry and from which he acquired in 1902 his Master's degree in Arts. Continued postgraduate research at the University of Pennsylvania (1904–1908) contributed to his degree of Master of Science from Ohio Wesleyan in 1910 and later (1926) to that of Doctor of Science from the same university.

Even before these four years of specialized investigation were concluded, the Philadelphia period of Doctor Snook's productive career may be said to have begun. For a short time, which included the year 1902, he was associated as a wireless-telegraphy expert with Queen and Company, scientific-instrument makers. His increasing interest in the further development of the induction-coil, as bearing upon the creation of powerful discharges for the ultimate and practical production of roentgen rays, gradually directed his abilities into the field of manufacture, in commercial application of the fruits of his creative effort.

To this end, but in the face of an industrial future as yet uncertain, even after eight years of the roentgen rays, Clyde Snook had the vision and the courage to form an organization for the fabrication of roentgen apparatus embodying his conception and design. This was known as the Roentgen Manufacturing Company of Philadelphia, established in 1903 and which later became the Snook-Roentgen Manufacturing Company. He was President of each.

The plan was sound, the opportunity proved to be ripe, enthusiasm among "x-ray workers" ran high and the Company prospered. Under these happy auspices materially augmented by the results of his high standards of production, the earlier types of Snook's excellent coils and interrupters were introduced and promptly acquired a fine reputation for design and construction. His engineering accomplishments in this specific field culminated in the development of his "Jumbo" coil, with electrolytic interrupter, which probably, at that day, represented the maximum of energy output for x-ray tube-exciters of this class. It was built with a notably thick core of the laminated type and with a "variable inductance" controlled at will by means of a plug-and-socket tapping-selector switch at one end of its primary encasement.

Despite the efficiency attained by the induction coil through his elaborations, Snook could not thereby entirely escape the two major obstacles attending its use—the phenomenon of inverse discharge and the annoyance entailed in the necessary employment of interrupters for the intermittent breaking of the primary circuit. The former problem especially he investigated thoroughly during 1906, reporting to the American Roentgen Ray Society his analysis of the causes of inverse discharge. This communication represents the background for the study and the experimentation which brought forth, in 1907, the permanent solution of these difficulties in the shape of the interrupterless transformer or "Snook Machine," as it was more familiarly known.

Among living American radiologists there may be those who remember the very earliest examples of Doctor Snook's apparatus, designed for more frequent use with the direct current that was so commonly supplied to urban laboratories at that time. Even in this initial and unimproved mecha-

nism, of but two-kilowatt rating, one saw the permanent banishment of the temperamental interrupter and no slight trace of inverse current that was not immediately corrigible. Its cross-arm rectifying switch rotated alone within a dust-proof housing; its high-tension transformer tank and the workings of its motor-convertor, on the other hand, were in large measure exposed to view. This apparatus was the precursor of all that was to follow, down through the years of alternating-current energization and disc-type rectification, of auto-transformer and filament-transformer, to the era of the kenotron valve.

In the bright light of this momentous product of Clyde Snook's genius, it is not remarkable that to the radiologist of normally elastic memory or of average historical inclination, his name may appear perhaps less vividly in connection with other notable results of his creative powers. How many today, for example, will recall that in gas-tube days he developed a hydrogen tube with dual osmotic regulation for the purpose of admitting or of dispelling this gas electrically, to "lower" or to "raise," respectively, the degree of vacuum?

A wholly indispensable instrument in daily use by the roentgen-ray technician and for which he is indebted to Doctor Snook is the milliammeter. Its development dates back to the induction-coil days of 1904, marking the modern period of exactitude in electrical mensuration for radiation purposes.

Among accessory devices that came into being through Snook's sustained energies was an upright fluoroscope having a complete radiopaque encasement for the tube and otherwise so carefully constructed that it offered thorough protection to patient and operator at a day when radiation danger was almost omnipresent. This apparatus was not easy of manipulation, however, and for this reason alone was not widely used. About the time of the first Great War, Snook introduced a new form of crossed-thread localizer, as well as a clever adaptation of the Sweet apparatus whereby are assured both complete immobilization and accuracy in adjustment.

To a degree unusual for a scientist not medically trained, Clyde Snook from the very beginning of his Philadelphia days formed and retained a close fellowship with medical men identified with roentgenology and its growth. He was a charter member of the old Philadelphia Roentgen Ray Society, that pioneer group assembled by the late Lester Leonard in 1906 and as Secretary of which he succeeded Doctor Leonard in 1913. Thus when, in 1916, the Snook-Roentgen Manufacturing Company was amalgamated with the Victor Electric Corporation and he removed to Chicago as Vice-President of the combined organization, it was to the infinite regret of all who had known him for so many years.

But Snook had no enduring fondness for the administrative work of a commercial corporation. He was in essence an individualist, born to the atmosphere of the laboratory of applied science. After two years opportunity came to him in a field that had long attracted him—that of sound transmission and the communication art, with especial reference to electronics. From 1918 to 1925 he was with the Western Electric Company, an ally of the Bell Telephone Laboratories, first as Staff Engineer, then in charge of its physical laboratory, which later became a part of the Bell organization under the continued direction of Doctor Snook, as Electrical Engineer, from 1925 to 1927. Since 1927 he had been practising as a Consulting Engineer. It was in 1926 that he produced his highly microphonic electrical multiple stethoscope which, in addition to its sensitivity, is capable of demonstrating bodily sounds to many ears. He was Chairman of the Noise Elimination Committee of the National Safety Council in 1930.

In addition to his collegiate acquisitions, which included Phi Beta Kappa and Phi Delta Theta, Doctor Snook received many honors coeval with his brilliant career. In 1919 he was awarded the Edward Longstreth medal of the Franklin Institute; in

1923, the gold medal of the Radiological Society of North America; in 1928, the gold medal, with honorary Fellowship, of the American College of Radiology. He was also a Fellow of the American Institute of Electrical Engineers and a Fellow of the American Physical Society. He was a Member of the American Roentgen Ray Society and a Charter Member of the Philadelphia Roentgen Society.

Anyone who has had even limited contact with the course of Clyde Snook's life will agree that the conspicuous threads in the stout fabric of his character were his modesty, his sincerity and his felicity. His modesty was expressed in his reaction toward any attempt improperly or unduly to publicize his accomplishments, One who was very dear to him and who knew him best has said that ". . . he cared little for publicity and only hoped in a quiet way to be able to contribute something from time to time to Science." How truly do such sentiments as these, voiced throughout the pages of Biography, reflect the philosophy of the scholar!

The sincerity in Doctor Snook's character was apparent in his love of truth—truth manifestated not only in human conduct, but as honesty in human handiwork. This was his fundamental specification for the concrete products of his engineering skill and, through his insistence, became the intangible label by which these things are known.

Felicity, with Clyde Snook, was no quality of mere personality. It was innate, and as such emanated from the chambers of his heart. It adorned his individualism and indicated its presence through his ready smile and his hearty laugh, whereby his medical friends were fascinated and their technical problems melted into thin air. For these and the rest of his splendid qualities he will be sorely missed by his former colleagues of the American Roentgen Ray Society, as well as by the survivors of the old Philadelphia group who wished him God-speed on that December evening twenty-six years ago and with whom, as he went, he left his heart!

Percy Brown

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual Meeting: 1943, to be announced.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual meeting: 1943, to be announced.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: 1943, cancelled.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Drake Hotel, Chicago, Ill., Nov. 30–Dec. 4, 1942.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. J. D. Coate, 434 Thirtieth St., Oakland, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGFLES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Donald R. Laing, 65 North Madison Ave., Pasadena, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N.Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave., Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. J. O. Newton, 13921 Terrace Rd., East Cleveland. Meets at 6:30 P.M. at Midday Club on fourth Monday each month, October to April, inclusive.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in April.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. J. P. Medelman, 572 Lowry Medical Arts Bldg., St. Paul.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. M. M. Pomeranz, 1020 Park Ave., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Ohio Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital. Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Francisco Radiological Society
Secretary, Dr. H. A. Hill, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months at Toland Hall, second at Lane Hall.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. L. W. Baird, Scott and White Clinic, Temple, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00-5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. C. H. Peterson, 603 Medical Arts Bldg., Roanoke, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. K. J. Holtz, American Bank Bldg., Seattle. Meets fourth Monday each month, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets monthly on third Thursday from November to June inclusive, at 8:15 P.M., 32 Welbeck St., London.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress.
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

ACKNOWLEDGMENT

To the Editor:

At this time, Dr. Lewis C. Sheffey has brought to my attention the following paper: "Adenocarcinoma of the cervix in a twenty-two months' old child" by Lewis C. Scheffey and Baxter L. Crawford, Department of Gynecology, Jefferson Medical College Hospital, Philadelphia, published in the *American Journal of Obstetrics and Gynecology*, 1932, vol. 24, pp. 118–122.

This reference was inadvertently omitted from the review of the available literature in the paper entitled: "Carcinoma of the cervix uteri in childhood and adolescence," by Harry H. Bowing and J. A. L. McCullough, Section on Therapeutic Radiology, Mayo Clinic, Rochester, Minnesota, published in the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY, 1941, vol. 45, pp. 819–826.

Scheffey and Crawford's report is of a proved case of adenocarcinoma of the cervix in an infant and is deserving of this belated acknowledgment.

HARRY H. BOWING

FLORIDA RADIOLOGICAL SOCIETY

The newly elected officers of the Florida Radiological Society are as follows: *President*, Dr. Elliott M. Hendricks, Fort Lauderdale; *Vice-President and Acting Secretary*, Dr. Walter A. Weed, Orlando; *Secretary-Treasurer*, Dr. J. Maxey Dell, Jr. (in Service), Gainesville.

NOTICE

Through an oversight in the obituary of Sir William Bragg which appeared in the May, 1942 issue of the JOURNAL, attention was not called to the fact that he was at one time President of the Royal Society and among the many honors which he received was the Order of Merit.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

ROENTGEN DIAGNOSIS OF THE EXTREMITIES AND SPINE. (*Ann. Roentgenol.*, Vol. XVII.) By Albert B. Ferguson, M.D., Formerly Director of Roentgenology, New York Orthopaedic Hospital, and Instructor in Orthopaedic Surgery, College of Physicians and Surgeons, Columbia University. Enlarged first edition. Cloth. Price, $12.00. Pp. 462, with 562 illustrations. New York: Paul B. Hoeber, Inc., 1941.

This volume is an enlargement of the first edition which appeared in 1939. In the reprinting, the author has expanded the chapter on the spine to include lumbosacral anomalies. He was stimulated to do this by the interest shown in the Lame Back Exhibit at the meetings of the American Medical Association. The section now contains thirty-five pages and fifty-five illustrations and approximates the detailed exposition offered in the Lame Back Exhibit. The other sections of the book are unchanged.

The first edition was well received in 1939 and was commented upon very favorably. In the arrangement of the text in both the original and reprinted editions, the author has consistently adhered to his belief that the differentiation and identification of common conditions is a topic sufficiently voluminous and important to be the sole subject of the entire text. Since exhaustive compilation which covers every phase of every lesion would demand a separate volume for each disease and each region of the body, he has confined the text to material of diagnostic utility. In the introductory chapter he defines the roentgenological nomenclature which he employs and he adheres strictly to this throughout the entire book. The specialized terms appear in bold face type and thus arrest the reader's attention. He emphasizes the necessity of the use of a terminology in the description of roentgen findings which excludes the haphazard, verbose and meaningless expressions so often employed.

Graduate students in roentgenology have found it difficult to use the first edition as a reference book because they are accustomed to arrangement of texts in the form which the author has deliberately avoided, namely, compilations of data on each disease and on each region of the body. However, when urged to regard the volume as a textbook of differential diagnosis and to view it from the standpoint of a critical and analytical examination of the individual lesions, they soon appreciate the wealth of material in the text, the uniform and consistent differential analysis of diagnostic criteria, and above all, they cannot but be impressed by the value of a specific, definitive terminology in the descriptions of the various roentgenological changes found in osseous lesions. The student, too, may find difficulty in appreciating the degrees of bone density which Ferguson believes are characteristic for lues, nonspecific inflammatory disease and neoplasms, or he will fail to perceive the difference in appearance between sclerosis, condensation and non-reticulated areas in bone as differentiated by Ferguson, or again, the different appearance of the various types of subperiosteal calcification. Careful reading and study of the book, however, will repay the reader and even though he may disagree that such individual differential characteristics do exist, he will benefit by attempts to base his diagnosis of bone lesions on the diagnostic criteria as defined by Ferguson.

The book covers the entire skeleton in health and disease, not only in the descriptive text but with the aid of five hundred and fifty-four illustrative roentgenograms and eight clinical drawings. The characteristics of normal and diseased bone are presented objectively in each chapter. At the end of the chapter a short clinical abstract is listed for each roentgenogram reproduced. The new chapter on the spine adds greatly to the value of the book. It should be included in the library of every roentgenologist and orthopedic surgeon and can be recommended most highly to every practitioner and student.

R. S. BROMER

THE AUTONOMIC NERVOUS SYSTEM: ANATOMY, PHYSIOLOGY, AND SURGICAL APPLICATION. By James C. White, M.D., Assistant Pro-

fessor and Tutor in Surgery, Harvard Medical School; Chief of Neurosurgical Service, Massachusetts General Hospital, Boston, and Reginald H. Smithwick, M.D., Instructor in Surgery, Harvard Medical School; Assistant Visiting Surgeon, Massachusetts General Hospital. Second edition. Cloth. Price, $6.75. Pp. 469, with 92 illustrations. New York: The Macmillan Company, 1941.

In the excellent Foreword of the book, Cannon remarks that "the opportunities of progressing beyond the boundaries of familiar surgery seemed to have been coming to an end." But that was true before Leriche in France, Adson and White and Smithwick in the United States started their impressive advances in applying surgical technique to the treatment of pathological states resulting from abnormal autonomic activity.

This present second edition of the book by White and Smithwick tells of their share in the developments in this field. It does more, because it is an ample book both in scope and thoroughness. The historical development of knowledge of the involuntary nervous system is followed by chapters on the anatomy and then general physiology. The latter chapter is particularly fine. It sums up the rôle of the thoracolumbar division as "an emergency protective mechanism, which may not be functioning all the time but which is always ready to go into action to combat any variety of adverse circumstance. Some of the most common conditions which arouse its activity are pain, extremes of temperature, asphyxia, hemorrhage, infection, dehydration and hypoglycemia. Furthermore, any form of intense emotion or psychic trauma may stimulate a generalized sympathoadrenal discharge. Cannon (1933) has emphasized the interesting fact that this mechanism may actually be harmful unless the emotion is transformed into action. . . . Little attention has been paid to this effect by the medical profession although it is of profound importance in human psychology." Again to emphasize the authors' comprehension of the clinical, one finds in the chapter on general physiology, "In health, visceral function is regulated so well by the autonomic nuclei in the hypothalamus that the normal individual is nearly unconscious of his internal organs. As Bard's work (1939) has shown, this regulating center is normally kept in check by the cerebral cortex but when upset in any way it may work erratically and thereby cause disagreeable symptoms in many organs of the body, viz., palpitation, vasomotor disturbances, insomnia and many varieties of gastro-intestinal disorders."

A brief review of such a book is impossible. Methods of study of the autonomic system are detailed and there is a full chapter on the drugs and hormones to be used both in study and therapy.

Then follows Part II in which a wide range of clinical conditions based on autonomic system disorders is described. In the section on peripheral vascular disease, the portion dealing with Raynaud's disease could not be improved.

Reading the section on extremities, bones, joints and pain makes one wonder if the orthopedic surgeon will not be led to add some surgery of the sympathetic system to his armamentarium.

The ophthalmologist will find a brief paragraph dealing with cervicothoracic ganglionectomy in retinitis pigmentosa, the urologist a chapter on the innervation of the urogenital tract and the internist no internal organ in his domain which cannot be modified in its performance by the neurosurgeon.

In fact, while for several years we have now been told that the future of medicine lies along the road of biochemistry, the promise of surgery in this new field makes one suspect that the pendulum is more apt to swing back to anatomy.

Certainly every neurologist should want this book and there is no one working in any field of medicine who would fail to gain a great deal of knowledge from its study. This praise is warwanted, because throughout, the book is characterized by historical thoroughness, research diligence and remarkably good clinical acumen.

Thomas K. Davis

Clinical Aspects of the Electrocardiogram Including the Cardiac Arrhythmias. By Harold E. B. Pardee, M.D., Assistant Professor of Clinical Medicine, Cornell University Medical College; Associate Attending Physician, New York Hospital; Consulting Cardiologist, Woman's Hospital and Methodist Hospital, etc. Fourth edition revised. Cloth. Price, $5.75. Pp. 434, with 102 illustrations. New York: Paul B. Hoeber, Inc., 1941.

Pardee's earlier editions of this now most comprehensive volume on electrocardiography long stood as a monument of clarity to the students of this science. The fourth edition

retains much of the clarity of the earlier editions, but has broadened in scope and grown in comprehension until it now constitutes a nearly complete reference work on the subject. The book is of extreme value to the advanced student of electrocardiography, for there is little of importance that is not amply covered; but for the younger physician certain portions are at times perhaps a bit too comprehensive and elaborate in electrical and physiologic detail. The portions of the book dealing with the precordial leads are extremely well covered as to practical information and bibliography. Pardee's broad experience is manifest in the splendid handling of such important subjects as the prominent Q3, low voltage QRS and T waves, and the problem of prognosis in relation to the electrocardiogram. While several well written treatises on electrocardiography have appeared in the past four years, this latest edition of Pardee's is well justified on its merit.

Warren B. Cooksey

Cancer of the Face and Mouth: Diagnosis, Treatment, Surgical Repair. By Vilray P. Blair, M.D., Sherwood Moore, M.D., and Louis T. Byars, M.D., St. Louis. Cloth. Price, $10.00. Pp. 599, with 260 text illustrations and 64 plates. St. Louis, Mo.: C. V. Mosby Co., 1941.

This volume is essentially a résumé of the surgical experience of one clinic where approximately 1,500 cases of carcinoma arising from the epithelium above the clavicle have been treated during a period of twenty years. A brief chapter on the general care of the cancer patient is followed by well illustrated sections, each of which deals with cancer of a single region. Tumors of the jaws and salivary glands, and moles, nevi and melanomas also receive adequate attention. The last 215 pages are devoted to carefully drawn sketches illustrating operative techniques and the details of plastic repair.

A short well written chapter on radiation therapy is compressed into nineteen pages and is accompanied by a good bibliography. Although the value of this method is rather grudgingly admitted in other portions of the book, the superiority of surgical procedures is implied if not openly claimed and the authors are much concerned over the growing enthusiasm of those "advocating pseudosurgical practices that are of less than questionable value" and "claims that cannot always be verified by unequivocal clinical experience." It seems unfortunate that they did not bolster this attitude by the inclusion of carefully compiled surgical statistics from their ample material for comparison with figures published by recognized radiological centers. An effort is made to partially rectify this defect by including the five year statistics compiled for 780 patients of the late Dr. Fischel but these figures are not arranged for a comparative study.

The clinical value of the book cannot be questioned but its usefulness is definitely marred by an obvious disregard for the work of others as evidenced by a dearth of references to the literature. The radiological chapter deals almost exclusively with palliative procedures and the treatment of the basal cell carcinoma. Radiation therapy in the treatment of metastatic cervical lymph nodes is condemned because of the poor results obtained with roentgen therapy alone and most of the case histories are characterized by a surgical boldness not matched by the radiological techniques, many of which are incompletely described. Although this volume contains a plea for better cooperation between radiologists and surgeons it really represents the best that surgery has to offer in cancer of the head and neck.

Charles L. Martin

Medical Progress *Annual* 1940: A Series of Fifty-two Reports Published During 1940 in the New England Journal of Medicine. Robert N. Nye, M.D., Managing Editor. Cloth. Price, $4.00. Pp. 625. Springfield, Illinois: Charles C Thomas, 1941.

One of the major problems in medicine is to provide some means whereby the physician may continue his education following graduation. Obviously, a modern teaching program is not devised to saturate a medical student with sufficient information to meet all of his needs for the remainder of his professional career. Not only would this be impossible because so much data could not possibly be assimilated by the student in four years, but because the science of medicine is fast moving and vitally essential information is being added to our sum total of knowledge each day. Furthermore, even the specialist who limits his chief interests to a sharply restricted field, finds it exceedingly difficult, despite continuous daily reading, to keep abreast with all of the new and important articles which should be perused. It becomes necessary, therefore, to provide some type of

critical review to meet the requirements of the medical profession. The collection of 52 articles dealing with a wide variety of medical and surgical subjects, which appeared in the issues of the *New England Journal of Medicine* during the year 1940, have now been bound in book form. They appear to be a sincere and earnest attempt to meet the needs of the advanced medical student, the practitioner, and the specialist.

This collection of reviews has much to commend it. First, the topics selected are well chosen, 16 being surgical, 27 medical and a number dealing with miscellaneous topics and the fundamental sciences, such as bacteriology, pathology, and physiology. Second, the authors who have prepared these articles are well qualified men who are capable of authoritative statements. Third, the subject matter is carefully selected, not entirely on the basis that the material is new, but also that it has been accepted and should be utilized. And, finally, they are short, to the point, and for the most part the material is presented in a terse, clear and understandable style.

This collection of reviews is to be commended to all who are desirous of keeping informed in regard to the advances in medicine. If each article were carefully read and digested, it is unlikely that any recent contribution of importance would be overlooked. These presentations also serve a very useful purpose as a basis for further study of the literature in any given subject.

Cyrus C. Sturgis

Tumores primitivos malignos bronco-pulmonares: Cancer, sarcoma, linfogranuloma. By Julio Palacio, Professor Adj. de Clinica Medica de la Fac. de Med. de Bs. Aires; Director del Dispensario Municipal de Vias Resp. No. 2, and Egidio S. Mazzei, Prof. Adj. de Clinica Medica de la Fac. de Med. de la Plata; Jefe de Clinica de la Catedra del Prof. Castex, etc. Paper. Pp. 401, with 129 illustrations. Buenos Aires: El Ateneo, 1940.

The authors' personal experience with 120 cases of primary malignant disease of the lung and the knowledge gained by a review of 1,200 references are presented here. Their own cases from the Catedra Clinica Medica of Prof. Dr. M. R. Castex of Buenos Aires were worked up completely from a clinical, roentgenologic, and in most cases also from a pathologic point of view, and the data are presented in a way understandable by those with even a minimal knowledge of Spanish. Particularly worthy of mention are some colored plates of pathologic and bronchoscopic specimens. Of interest to radiologists is the presentation and correct evaluation of the various roentgenologic procedures and their findings. The authors offer nothing new in way of treatment for bronchiogenic carcinoma. Sarcoma of the lung, a very rare lesion, was encountered twice in the authors' series.

This monograph is a noteworthy contribution to the better knowledge of malignant pulmonary tumors, a subject of growing interest, importance and complexity and merits serious consideration by clinicians, surgeons, radiologists and pathologists, all of whom can well derive profit from this book.

E. T. Leddy

Estudio radiológico de la equinococcosis pulmonar. By Manuel Arce and Francisco Arce, Jefes del Servicio del Departmento de Radiodiagnóstico del Hospital Asilo de San Rafael, Hospital del Niño Jesus y Clínica de Pediatría de San Carlos. Colección Española de Monografías Médicas. Director: Dr. J. Puig Sureda. Paper. Pp. 75, with 36 illustrations. Barcelona: 1941.

The authors, who are well known for their work on various intrathoracic diseases and particularly for their bronchographic studies of them, report here some important observations they have made on hydatid disease of the lungs. Even though the lesion is uncommon in this country at present, it may be that soldiers and sailors returning after the war may present this lesion with sufficient frequency to necessitate serious consideration of it in roentgenologic differential diagnosis. In this volume the authors present the fundamentals of bronchography as it applies to this lesion; they then discuss closed hydatid cysts and their complications and their treatment, the various causes and types of rupture of the cyst into the bronchi, secondary manifestations due to sequelae or to residual changes, which produce suppuration or break down of tissues, and then echinococcosis of the pleura, multiple primary hydatid disease and echinococcal infection of the base of the lungs. There is a bibliography of 106 references. This book deserves study by all roentgenologists and others who may be actively concerned with diseases of the lungs.

E. T. Leddy

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

GYNECOLOGY AND OBSTETRICS

CHAPPLE, C. C., and DAVIDSON, D. T. A study of the relationship between fetal position and certain congenital deformities. *J. Pediat.*, April, 1941, *18*, 483–493.

Uterine pressure is accepted as a cause of certain congenital deformities. The authors feel that the exertion of this pressure depends on fetal posture in part. By studying the attitude of the fetus as a whole they were able to trace the mechanism of the production of certain deformities. Their interest in the subject was stimulated by an infant born with marked lateral bowing of the femurs. In order to explain such a deformity they postulated uterine pressure as a mesially directed force and some laterally directed force at the apices of the concavities of the bowed femurs to act as a fulcrum. To find such a fulcrum they folded the child's legs tailor-fashion so that each leg met the opposite femur at right angles. To their surprise the child was perfectly contented in this cramped position and objected to any attempt to alter it. They call this the "position of comfort." This indicated that the child had become accustomed to this position in utero and further confirmation was found in the roentgenogram which showed lateral dislocation of the hips which could only result from a laterally directed force.

With this first case as a stimulus they made routine studies on newborn infants and were able to uncover deformities which had not been suspected. They feel that their theory offers an excellent explanation for the production of congenitally dislocated hips. Pressure is necessary for the conversion of the cartilaginous acetabulum and femoral head into bone. The integrity of the hip joint is dependent mainly on the joint capsule even in normal fetal position. Dislocation might occur suddenly with normal acetabula due to excessive uterine pressure and yielding of an over-strained capsule and complete dislocation occur. In many cases, however, a relaxed capsule is present and roentgenograms show delayed ossification of the acetabula and delayed appearance of the femoral capital epiphysis. The authors suggest capsular relaxation may be due to the relaxing hormone of the mother.

Dislocations were also present in the knee joints and rarely in the shoulder, elbow and hip joint.

The authors include illustrations of several unusual and interesting "positions of comfort" and diagrammatic illustrations of the roentgen findings in congenital dislocation of the hips. They feel that determination of the "position of comfort" should form a part of the routine examination of the newborn in order to explain existing deformities and, more important, to anticipate and prevent possible deformities.—*R. M. Harvey.*

GENITOURINARY SYSTEM

DANELIUS, GERHARD. Kidney stones simulating biliary concretions. *Radiology*, March, 1941, *36*, 324–333.

Very little attention is paid in the roentgen literature to the fact that kidney stones may be mistaken for stones in the biliary tract. The author describes and gives roentgenograms of 3 such cases. The first patient was a woman of thirty-six who had symptoms of osteomyelitis, the second a man of twenty-seven who had no symptoms except vague pain on the left side and the third a very obese woman of forty-five who had the typical clinical picture of cholecystitis which was found on operation to be caused by a strawberry gallbladder and cholecystitis. None of these patients had any urinary symptoms and in all of them the roentgen picture was typically that of a gallbladder filled with stones, except that in the second case the stones were on the left side and examination showed that there was no situs inversus. In all the cases further study showed the stones in the kidney.

Laminated kidney stones, which are the ones most apt to be confused with biliary stones, may be formed in calyces with defective drainage when there has been infection of any kind. There was no evidence of tuberculosis in any of the cases described.—*Audrey G. Morgan.*

Pierson, L. E., and Honke, E. M. Unilateral fused kidney with bilateral ureteral bifurcation. *J. Urol.*, May, 1941, *45*, 682–684.

Over 300 cases of unilateral fused kidney have been reported in the literature. The authors report a case in a woman, aged fifty-eight, who had persistent pyuria. Survey films of the abdomen were exposed with opaque catheters in each ureter. The left catheter was found to lie laterally to its normal course and the right ureter crossed the midline over the upper sacrum. Retrograde pyelograms showed bifurcation of the left ureter at the junction of the middle and lower thirds. The right ureter was also bifurcated. Five of the cases described in the literature of unilateral fused kidney had bifurcated ureters. The authors' case is the first reported case discovered antemortem.—*R. M. Harvey.*

Boyd, M. L. The significance of sensory paralyses in poliomyelitis patients in whom renal calculi develop. *J. Urol.*, May, 1941, *45*, 647–651.

The author calls attention to the obscuring of symptoms in the presence of renal calculi in poliomyelitis patients due to sensory changes. Urinary infections are common in paralysis cases and factors are present which lead to stone formation. It is therefore very important to take measures to prevent stone formation, clear up urinary tract infections when they occur, and make regular routine survey films of the abdomen to detect the presence of calculi which do not make themselves evident by the usual symptomatology. The fluid intake should be adequate, acidification of the urine should be maintained and frequent changes of position of the patient should be made.

The author quotes the case of a doctor's son who developed an obscure fever following the onset of paralysis. He did not complain of urinary symptoms, however, and the fever was attributed to his poliomyelitis. When an examination was finally made of the urinary tract the patient was found to have a large stone in the left ureter with long-standing chronic pyelonephritis.—*R. M. Harvey.*

Alexander, J. C. Pneumopyonephrosis in diabetes mellitus; case report. *J. Urol.*, April, 1941, *45*, 570–578.

The author reviews the literature on pneumaturia, meaning air or gas in the urine. Senator divides pneumaturia into 3 classifications: (1) atmospheric air introduced by cystoscopy or catheterization; (2) fistulous air from connection of the urinary tract with a hollow viscus, and (3) fermentation by the action of organisms on glucose-containing urine. The author is concerned with the latter classification.

His patient was a female diabetic, aged sixty-three, who was admitted complaining of left flank pain, frequency, chills and fever, and with the history of having passed a small stone. A scout film of the abdomen showed a spontaneous air pyelogram of the left kidney. At cystoscopy the left ureter could not be catheterized due to edema. Later thick foul urine was obtained from the left renal pelvis. No gas bubbles escaped from the catheter which was anchored in the left kidney for drainage. The patient's general condition became worse and a second scout film of the abdomen showed gas surrounding the entire left kidney. The capsule was separated from the renal cortex at the lower pole for at least 1 cm. An attempted retrograde pyelogram showed that the gas had extravasated from the kidney pelvis under the capsule. At operation the capsule ruptured and gas and serosanguineous fluid escaped. This fluid showed colon bacillus and *Staphylococcus albus* on culture. A fecal odor was present.

The author feels that instrumentation is contraindicated where scout films show the presence of pneumaturia and that immediate nephrostomy or nephrectomy should be performed.—*R. M. Harvey.*

Rose, D. K. Influence of psoas major muscle on kidney function. *J. Urol.*, April, 1941, *45*, 558–569.

The psoas major muscle is important because through its relation to the ureter and kidney pelvis it may cause urine stasis and secondary functional alterations in smooth muscle. These alterations vary from hyperplasia to complete decompensation. The author found at the dissection table that the relation of the kidney to the psoas major muscle presents certain variants. These are (1) variations in size of the psoas shadow according to habitus of the individual; (2) variations in relation of position of kidney and ureter to the psoas muscle; (3) variations in course of ureter in gaining its position on the muscle, and (4) variations in the position of the hilum in relationship to the inner border of the kidney, whether anterior or pos-

terior. Posterior rotation is especially important since it might force the ureter to climb upward to reach the anterior surface of the muscle.

The author feels that contraction of the psoas shadow may cause interruption of normal ureteral peristalsis. In the presence of a thick psoas and a mobile kidney the ureter must climb over the psoas belly and this could prevent descent of a calculus. Furthermore heavy connective tissue bands may form at these points of physiologic interference with ureteral function. All these stasis factors have importance in infection, stone formation, and pain or back pressure damage to renal function. To bring out these points intravenous urograms without abdominal pressure and with erect films are necessary.

Illustrative urographic films are shown showing various effects on the abdominal ureter, especially at the ureteropelvic junction by variations in the relation between kidney, ureter and psoas muscle.—*R. M. Harvey.*

BENJAMIN, BEN, and RATNER, MAX. Hypertension associated with unilateral chronic atrophic pyelonephritis; occurrence in a child in whom no decrease of blood pressure followed nephrectomy. *Am. J. Dis. Child.*, May, 1941, *61*, 1051–1063.

Benjamin and Ratner report the case of a six year old boy who had hypertension associated with pronounced atrophic pyelonephritis, and marked functional impairment of one kidney and no evidence of any other abnormality. He was subjected to nephrectomy. There was no decline of the systolic or the diastolic blood pressure immediately after the operation or during the following six months. Retrograde bilateral pyelogram showed a relatively normal right renal pelvis and ureter and a markedly distorted left renal pelvis with absence of calyces. They review the subject briefly to emphasize the inadequacy of the present knowledge of the etiologic relationship between hypertension and nephropathy. In their case, it appeared reasonable to expect a favorable result. They conclude that the relation between hypertension and renal disease requires further study and elucidation. It appears impossible at present to formulate definite criteria for the selection of cases of unilateral nephropathy and hypertension in which it may be expected that nephrectomy will be followed by a reduction of blood pressure in every instance. For any child who has chronic hypertension associated with renal disease a urologic examination is advisable. If the examination clearly demonstrates the presence of a unilateral renal lesion and there is no contraindication to operation, nephrectomy may be justified.—*R. S. Bromer.*

STANG, H. M., and HERTZOG, A. J. Primary carcinoma of the ureter. *J. Urol.*, April, 1941, *45*, 519–526.

The number of cases of primary carcinoma of the ureter reported to date, including the authors' cases, now totals 155. Secondary tumors of the ureter are usually implantations from papillary renal carcinomas. Primary tumors arise from transitional epithelium and are squamous cell type, as a rule. They may be flat or papillary.

The authors report 4 cases. In each case pyelography showed ureterectasis and pyelectasis. All 4 cases were histologically squamous cell carcinoma; 3 cases were in elderly men and 1 in a young woman. One case survived four years without evidence of recurrence. The usual complaints were pain and hematuria. Cystoscopic examination often reveals these tumors projecting from the ureteral orifice. The differential diagnosis includes renal calculi, neoplasms of the renal pelvis and bladder and tuberculosis.

The prognosis is very poor. In a series of 62 cases the operative mortality was 27 per cent and only 2 cases were well five years postoperatively. The treatment is complete nephroureterectomy. Irradiation postoperatively is of value in relieving the pain of metastatic lesions. —*R. M. Harvey.*

LAZARUS, J. A. Metastatic ureteral obstruction following carcinoma of the ovary; report of an unusual case of pyonephrosis resulting from a metastatic carcinoma of the ureter. *J. Urol.*, April, 1941, *45*, 527–535.

Direct extension of cancer of the cervix, prostate and bladder to the ureter is common but true metastasis by way of the blood stream or lymphatics is rare. The author's criteria for metastasis includes establishment of a primary malignant focus elsewhere, similarity of the ureteral tumor cells to the original tumor, and microscopic demonstration of the malignant cells in the lymphatics or blood vessels of the ureteral wall. The author summarizes 17 cases from the literature and adds 1 of his own. The primary lesion in 7 of these cases was in the prostate. The bladder, cervix and breast were

the primary sites in 2 cases each, and the bronchus, stomach, urethra and ovary were the sites in single instances.

His case was a female, aged sixty-eight, admitted with signs of a severe unilateral kidney infection which at operation was found to be secondary to a metastatic lesion of the ureter. The primary tumor was a squamous cell carcinoma arising within a dermoid cyst of the ovary. The symptoms of this lesion are those of any obstructive ureteral lesion.

In view of the common occurrence of primary tumors of the pelvic organs and the rich lymphatic anastomosis between these organs and the ureter it is difficult to understand why metastatic lesions of the ureters are not more common. The author does not feel that the downward flow of lymph in the ureteral wall is the explanation since no cases of ureteral metastasis secondary to kidney tumors have been reported.—*R. M. Harvey.*

Giuntoli, Lorenzo. Corpo estraneo di cera in vescica. (Wax foreign body in the bladder.) *Radiol. med.*, April, 1941, *28*, 234–235.

The author describes the case of a man, aged twenty-five, who complained of continuous suprapubic pain. Roentgen examination showed a reniform body of slight opacity in the bladder. It proved to be a mass of wax which looked like a candle bent on itself by a temperature which had been high enough to soften but not to melt it. The patient said he had had a gonorrheal urethritis for which medicated candles had been inserted into the urethra. At the same time the physician was giving urethral dilatations with a Benniquet sound. Apparently the wax had been pushed through the posterior urethra by the sound. One case of a wax foreign body in the bladder has been described previously.—*Audrey G. Morgan.*

Loeb, M. J. A case of adrenal cortical tumor without endocrinological symptoms. *J. Urol.*, May, 1941, *45*, 785–793.

Adrenal tumors arise from the cortex, medulla or their innervating sympathetic nerves. The cortex is mesodermal in origin and its tumors are therefore epithelial. The medulla is ectodermal in origin and, like the sympathetic nervous system, arises from the neural crest. The medulla is a part of the chromophil system and contains discrete masses of tissue which secrete adrenalin. Other parts of this system are the paraganglia of the sympathetic nervous system, the collateral ganglia of the abdominal region including the aortic bodies, and the carotid bodies and glomus coccygeum. Cells of this system contain granules which stain yellow or brown in the presence of chromium salts.

The cortical system consists of the cortex of the adrenal gland, and accessory cortical bodies near the ovary or testis. The cortex secretes a hormone essential to life. An excess of this hormone causes an adrenal genital syndrome, consisting of sexual precocity, pseudohermaphroditism, and changes in sexual character after maturity, as virilism. The syndrome called hirsutism consists of endocrine imbalance, pigmentation, obesity, hypertrichosis, muscular weakness, male voice, cessation of menstruation and psychic disorders. Hyperplasia of the cortex in intrauterine life results in pseudohermaphroditism. In males feminization and homosexuality occurs. Some cortical tumors show evidences of malignancy without the endocrine disturbances.

Ewing classifies cortical tumors as hyperplasias, adenomas and carcinomas. Geschickter states that benign cortical adenomas are found in one-third of all postmortems. Carcinomas of the cortex are very malignant, locally invasive and metastasize early.

The case which the author presents was that of a woman, aged forty-eight, complaining of weakness and weight loss for two years. Physical examination revealed a large abdominal mass. A survey film of the pelvis showed downward and inward displacement of the right kidney by a large mass. Bilateral pyelograms showed the upper pole of the right kidney compressed downward and rotated. Perirenal insufflation showed a retroperitoneal mass above and apart from the right kidney. At operation a tumor weighing 1,100 grams was removed, which histopathologically was a hypernephroma of the adrenal cortex. The patient was given postoperative roentgen therapy and rapidly gained in weight and strength. Eighteen months after operation she was apparently in good health. She had regained 40 pounds and her blood pressure was normal.—*R. M. Harvey.*

Halpert, B. Mixed leiomyoma and lymphangioma of the epididymis; report of a case. *J. Urol.*, April, 1941, *45*, 536–538.

Only 54 primary malignant tumors of the epididymis have been reported and 22 benign

tumors. The author reports a case of mixed leiomyoma and lymphangioma, a tumor which has never been described before. His patient was a colored male, aged sixty, who was admitted for prostatectomy. On discharge a swollen epididymis was noted and the patient returned later for its removal. The histological sections showed smooth muscle tissue and lymph capillaries embedded in a connective tissue stroma. —*R. M. Harvey.*

Huffman, L. F. Interstitial cell tumor of the testicle: report of a case. *J. Urol.*, May, 1941, *45*, 692–698.

The author summarizes the 9 previously reported cases of testicular tumors arising in the interstitial cells of Leydig and adds a case of his own. They are usually benign, encapsulated and yellow or brown in color. They give a negative test for prolan. Precocious development of the body and genitalia have been reported in some cases. The interstitial cells of Leydig are epithelioid cells of connective tissue origin and are found in the spaces between the seminiferous tubules. Their number varies with the individual. They are numerous in fetal life, decrease from birth to puberty and then increase until adult life. They are large polymorphous cells with abundant acidophilic cytoplasm containing lipoid and pigment.

The author's case was a boy of six years. He had a moderate growth of pubic hair and slight enlargement of both breasts. The left testicle was twice the normal size, firm, smooth and slightly tender. Friedman test was negative. Roentgen films of the bones were negative. Orchidectomy was done and the pathological diagnosis was interstitial cell tumor of the testis. Two years later the boy showed normal development although pubic hair and enlarged breasts persisted.—*R. M. Harvey.*

Ormond, J. K., and Prince, C. L. Malignant tumors of the testicle. *J. Urol.*, May, 1941, *45*, 685–691.

Among 319,000 men, women and children treated at the Henry Ford Hospital in twenty-six years there were only 21 malignant testicular tumors. Swelling and pain are the commonest symptoms. Other complaints are due to the occurrence of metastases. Length of history does not parallel tumor size. The majority of tumors in the authors' series were smooth and not very large. They did not find hydrocele to be a common occurrence with the tumor.

Metastases occur along the spermatic artery lymphatics to the retroperitoneal nodes, and eventually to the spermatic, epigastric, mediastinal and supraclavicular nodes. Metastases to the lungs may occur through the spermatic vein. The differential diagnosis lies between syphilis, tuberculosis, hydrocele, hematocele and benign tumor. Cryptorchidism appears to have a definite relationship to the disease but occurred only once in the present series. Single type cells and mixed cell tumors are found.

The five accepted methods of treatment are (1) orchidectomy; (2) deep roentgen therapy; (3) orchidectomy plus deep roentgen therapy; (4) orchidectomy plus pre- and postoperative roentgen therapy, and (5) radical orchidectomy with removal of the draining lymphatics. The most effective is a combination of orchidectomy and roentgen therapy.

In the reported series of cases 10 had no evidence of metastasis when first seen and of these 10 cases, 50 per cent survived more than four years. The remaining 11 had demonstrable metastases when first seen and all but 3 who could not be traced were dead within two years.—*R. M. Harvey.*

SKELETAL SYSTEM

Coste, F., and Gaucher, M. La sciatique dite "rheumatismale" a-t-elle vécu? Sciatique et arthrose lumbosacrée. Facteurs non-vertébraux des sciatiques. Conséquences thérapeutiques. (Is there such a thing as rheumatic sciatica? Sciatica and lumbosacral arthritis. Non-vertebral factors in sciatica. Conclusions in regard to treatment.) *Presse méd.*, Sept. 23, 1941, *49*, 1012–1016.

There has been an increasing tendency to attribute sciatica to posterior hernia of the intervertebral disc, particularly since reports have been made by American authors of a large number of successful operations in cases due to this cause. The authors believe, however, that while a certain number of cases of sciatica are due to this cause and certain others to repeated slight trauma causing compression of the disc without going so far as to cause hernia, the question is involved and more frequently there are a number of factors in the etiology and even though the old designation of rheumatic sciatica is perhaps to be rejected, nevertheless cold and dampness and exposure are factors in the causa-

tion of this condition. Rest with the patient lying down is still a valuable element in treatment. Further methods of treatment are antipyrine, salicylates and roentgen irradiation. The diagnosis of hernia of a disc as the cause of the condition can be made by lipiodol examination and if herniated disc is found laminectomy is indicated.—*Audrey G. Morgan.*

Delmas-Marsalet, P. Poussées évolutives gravidiques et image lipiodolée caractéristique des hémangiomes médullaires. (Case of hemangioma of the spinal cord with characteristic lipiodol picture and exacerbation during pregnancy.) *Presse méd.*, Sept. 10–13, 1941, *49*, 964–965.

A woman twenty-six years of age at the time of her second pregnancy in 1937 came for examination on account of pain in the arms and difficulty in walking. She had had the same symptoms beginning with the second month of her first pregnancy eight years before, but they had disappeared completely after the child was born. They had reappeared with the second month of the present pregnancy. This time again they disappeared after the delivery of a normal child. But in December, 1940, the pain began again without pregnancy and this time did not show any tendency to decrease. The pain was a characteristic nerve-root pain and there were also disturbances of sensation. The fact that there had been exacerbations during both pregnancies suggested that it was an angioma of the spinal cord and lipiodol examination was made. The lipiodol outlined tortuous vessels on the surface of the spinal cord extending from the second to the seventh dorsal vertebra. Laminectomy showed that the tumor was inoperable; the wound was closed and roentgen treatment given.

This case suggests that such nerve-root pain with exacerbation during pregnancy indicates a diagnosis of angioma of the spinal cord. A similar case has been described in which hemangioma of a vertebra was found, but the roentgenogram in that case had shown osteoporosis of the affected vertebrae.—*Audrey G. Morgan.*

Weinbren, M. Tomography of the spine and the sternum. *Brit. J. Radiol.*, Oct., 1940, *13*, 325–336.

A survey of the medical literature and visits to various hospitals have shown that tomography is used chiefly in examinations of the chest. Even here some writers claim that its value has been exaggerated but there is no doubt it has come to stay. The author finds, however, that tomography may be of great value in demonstrating fracture or infection of the spine or sternum in cases in which diagnosis from routine films has been doubtful. It is useful in differentiating between slight degrees of kyphosis resulting from slight compression fractures and adolescent kyphosis and in demonstrating the degree of consolidation in fractured vertebrae, showing lack of complete consolidation in cases that appear completely consolidated on the ordinary film. The position of intervertebral discs which have been forced into the bodies of the vertebrae by trauma may be demonstrated and these herniated parts of discs may explain certain symptoms. The best means of demonstrating the extent of callus formation in fractured vertebrae is by tomography. It may be used to make a differential diagnosis between old fractures and caries and between infection and injury. The best way to demonstrate the extent of tuberculous caries is by tomograms. Secondary deposits from the breast have been demonstrated by tomography when routine roentgenograms did not show them. The sternum is very hard to demonstrate by routine roentgenography and tomography has been found useful in demonstrating various pathological conditions in the sternum.

The greater part of the article is devoted to tomograms illustrative of the various conditions discussed.—*Audrey G. Morgan.*

Morris, Hugh. Suprasternal bones demonstrated in the living subject. *Brit. J. Radiol.*, Oct., 1940, *13*, 356.

A case is described in a man of thirty-two who came for examination for arthritis of the left ankle. But he complained of vague pains in other locations, particularly the sternum of which roentgenograms were made. They showed two suprasternal bones.

These bones are found in about 3 per cent of autopsy material but are rarely seen in the living subject. They vary in size from that of a grain of barley to that of a hazelnut and are of the same structure as the sternum with which they are fused in about 60 per cent of the cases. They occur as isolated anomalies, not associated with cleft sternum or cervical rib.—*Audrey G. Morgan.*

Horwitz, Thomas. Monomelic medullary osteosclerosis of unknown etiology. *Radiology*, March, 1941, *36*, 343–351.

A case is described of a benign bone lesion which, so far as the author has been able to determine, has not been described before. The patient was a woman of twenty-seven who came for treatment for a sciatica on the right side. Neither this nor the subclinical scurvy that was found on examination appeared to be related etiologically to the bone change found on roentgen examination.

The bone change was limited to the pelvis and left lower limb, that is to the derivatives of the left hind limb bud, the ischium, pubis, femur, patella, tibia and astragalus. There was no deformity or shortening of the affected bones and the joints were not involved. There was a diffuse osteosclerosis, involving chiefly the medullary region of the bones. In some places the cortex was found thickened from the inner side but there was no evidence of involvement of the periosteum. The normal spongiosa had been replaced by densely sclerotic bone. The newformed bone consisted of irregular segments of immature and adult bone arranged in curious forms.

This lesion is thought to be due to a developmental disturbance of the affected hind limb bud but its exact etiology is not known.—*Audrey G. Morgan.*

Reeves, Robert J., and Baylin, George J. Osteochondrodystrophy (Morquio). *Radiology*, March, 1941, *36*, 362–366.

Morquio in 1929 described an unusual picture of familial chondrodystrophy with dwarfism, kyphosis, genu valgum, pes planus and flabbiness of the muscles. Since that time some 30 cases have been described.

The authors report 2 cases in a brother and sister. The boy, five years of age when first seen, was said to have had "dislocated hips, knees and ankles" since he was one year of age. He could not support himself without crutches and his muscles were flabby. Laboratory studies were normal. There was no disturbance of calcium and phosphorus metabolism as reported by Morquio. Roentgen examination showed bilateral dislocation of the hips, delayed growth of the epiphyses, retarded development of all the carpal and tarsal bones and flattening and broadening of the distal diaphyses of the long bones, especially the femurs. The epiphyses were very ragged and of irregular density. This patient was followed up from 1931 to 1940. In 1937 he was treated for stricture of the esophagus. The skeletal deformities are now quite marked and the chest is distorted by the extreme kyphoscoliosis. His sister, a year older, presents practically the same symptoms.

These cases are presented in order to stimulate a further study of the cause of the disease. —*Audrey G. Morgan.*

Turpin, R., and Coste, F. La polyostéochondrite. (Polyosteochondritis.) *Presse méd.*, Oct. 21, 1941, *49*, 1136–1139.

Two cases are described in a father aged forty and a son aged ten. Both showed the same bone deformities, consisting of flattening of the bodies of the vertebrae, resulting in short stature and flattening of the heads of the femurs. The father had had difficulty in walking since childhood and his hands and feet were out of proportion to the other bodily measurements, but he had been able to perform his military service. The picture was that of an old osteochondritis.

The authors hold that this is the same disease that, in individual bones, has been called by as many as 30 names of men who have first described the condition in that particular bone, such as Calvé's disease of the hip, Köhler's disease of the scaphoid, Kienböck's disease of the semilunar bone, Friedrich's disease of the sternal end of the clavicle, etc. In all cases it is a growth injury of the bones involved developing on the basis of some constitutional predisposition, the exact nature of which is not known. Though this point is not emphasized in the literature it is often familial. Cases are cited from the literature. In the systematic examination of the skeletons of 100 couples the authors found that 17 of them had roentgen signs of osteochondritis, though none of these 34 persons complained of any symptoms.

This polyosteochondritis, due to a disturbance of ossification of the growth cartilages, is connected by various transition forms with the various forms of juvenile osteochondritis.—*Audrey G. Morgan.*

Lucherini, Tommaso, and Giacobini, Elio. Osteocondropatia universale familiare endocrina. (Familial generalized endocrine osteochondropathy.) *Radiol. med.*, March, 1941, *28*, 141–167.

Four cases of the above disease are described in the children of one family—two brothers and two sisters. The patients were twenty-one, seventeen, sixteen and fifteen years of age. They belonged to a family of seven children, the first, second and seventh of whom were not affected. There was no family history to explain the disease. There were no malformations in the family and no history of syphilis or tuberculosis. One grandfather was a drunkard.

Enlargement and flattening of the joints had begun in one of the patients at the age of three and in the others at the age of seven; that is, at those ages the deformity had become great enough so that the parents noticed it. It had gradually increased in all of them until at the time of examination all the joints were affected. The patients all had the appearance of adults and a characteristic depression of the bridge of the nose. The trunk was slightly flexed forward as a result of flattening of the bodies of the vertebrae; the thorax and spinal column were deformed so that the trunk was short in proportion to the length of the limbs. The gait was uncertain, with moderate valgism of the legs and exaggeration of the normal lumbar lordosis due to malformation of the bodies of the vertebrae and changes in the heads of the femurs.

Though the changes were in the epiphyses these could not be called cases of epiphysitis, for there were no evidences of inflammation. Differential signs from the usual forms of bone disease in young subjects, such as Köhler's disease, Kienböck's disease, Calvé-Perthes' and Ribbing's disease are given.

In addition to the joint changes all the patients showed unusual size of the sexual organs and early development of secondary sex characters and also precocious mental development. In 2 of the cases there were changes in the electrocardiogram indicating myocarditis.

The author concludes that this disease was probably caused by some abnormality of the hypophysis. The secretion of the anterior lobe of the hypophysis is the growth hormone, and abnormal sexual development is caused by dysfunction of this gland. There were digital impressions and diastasis of the sutures, especially the lambdoid suture in 3 of the patients indicating possible hydrocephalus and resultant injury of the hypophysis. Lumbar puncture was not permitted.

None of the treatments used so far, including extracts of hypophysis and parathyroid, mineral and vitamin treatments, short waves and roentgen irradiation have had any effect.—*Audrey G. Morgan.*

Thomas, H. W., Meredith, T. N., and Wunderly, H. L. Osteodystrophy disseminata. *J. Pediat.*, May, 1941, *18*, 638–642.

Three distinct types of pathologic conditions of bones, in which widespread rarefaction with cyst formation occurs, are recognized and may be differentiated through metabolic studies. The types are hyperparathyroidism, renal rickets and osteodystrophia disseminata. All show histopathological changes characteristic of osteitis fibrosa cystica. The non-protein nitrogen is elevated in renal rickets and normal in the other two; the blood calcium is elevated in hyperparathyroidism and normal in the other two, the fibrocystic changes are generalized in renal rickets and hyperparathyroidism and localized in osteodystrophia disseminata; the serum phosphatase is high in all three; urinary calcium excretion is high in hyperparathyroidism and intestinal calcium excretion high in the other two; skin pigmentation is present in only osteodystrophia disseminata in which sexual precocity occurs.

Osteodystrophia disseminata was first recognized as an entity in 1931. It is characterized by multiple areas of osteitis fibrosa cystica limited to one side of the body. Sexual precocity and pigmentation help to differentiate the disease.

The authors report a case of osteodystrophia disseminata in a male, aged three, with cystic changes in the right femur, tibia, maxilla and ilium. Blood calcium and phosphorus determinations were normal. The left testicle was enlarged and also the left breast. Deep areas of pigmentation were present over the left buttocks and sacrum. The urinary androgen was normal. No surgical attempt was made to determine the condition of the parathyroids.

Albright suggested a neurologic or embryologic origin of the disease because of the regional distribution of the lesions. A further point which may be of etiological significance is the preponderance of lesions on the right side. In the single case in which the lesions were left-sided the patient was left-handed.—*R. M. Harvey.*

Blewett, John. Cases of spondylolisthesis of unusual interest. *Brit. J. Radiol.*, Dec., 1940, *13*, 416–421.

Spondylolisthesis is an anterior displacement of the body of one vertebra on the body of the

one below it. This slipping forward is made possible by a double defect in the posterior arches of the displaced vertebra (spondylolysis). Two cases are described, one in a man of thirty-five and the other in a man of forty-five. In both cases the spondylolisthesis was of Grade 1 according to Meyerding's classification. The important point about these cases from the roentgen point of view was that the deformity could only be demonstrated when the patient was standing and could not be seen when he was lying down. Both patients were comfortable when lying down and felt the severe sciatic pain only when standing. The pain may be caused by the movement of the one vertebra on the other, or it may be caused by irregularities or retropulsions of the nucleus pulposus of the compressed intervertebral disc. Both these patients showed mental symptoms with a tendency to suicide but this may have been coincidence as no definite connection between the psychic state and the spondylolisthesis was demonstrated.—*Audrey G. Morgan.*

Huet, P., and Huguier, J. Syndrome radiologique de face au cours des luxations rétrolunaires du carpe. (Roentgen syndrome in the anteroposterior roentgenogram in retrolunar luxation of the wrist.) *Presse méd.*, Aug. 6–9, 1941; *49*, 860–863.

Examination of 34 unpublished cases of retrolunar dislocation of the wrist showed that in 17.6 per cent of the cases the correct diagnosis was not made on the first examination of the roentgenograms either by roentgenologist or surgeon. One case was diagnosed by a number of physicians and roentgenologists as a fracture of the scaphoid, a lesion which frequently accompanies retrolunar dislocation of the carpus. On lateral roentgenograms it is often difficult to diagnose the retrolunar dislocation on account of superposition of bone shadows, but on the roentgenogram taken from in front a characteristic triangle appears. The base is upward and convex, the pointed apex downward. It is the shadow of the lower surface of the semilunar bone which has been forced into a position of supination by the anterior radio-cubito-lunar ligament so that the lower surface looks forward and outward.

Photographic reproductions of tracings of roentgenograms in 35 cases are given.

This triangular appearance of the semilunar bone is characteristic of a double rotation around a transverse and a vertical axis. It may, however, be seen in traumatic antelunar luxations of the carpus and in pathological antelunar subluxation of the carpus in tuberculous osteoarthritis.—*Audrey G. Morgan.*

Giordano, Giovanni. Gravi deformazioni rachitiche. (Severe rachitic deformities.) *Radiol. med.*, May, 1941, *28*, 286–287.

The author gives roentgenograms of extremely severe rachitic changes in a patient, now a man of thirty, who had had rickets since early infancy and had not been treated.

The first roentgenogram shows a marked kyphoscoliosis of the whole spine accompanied by deformities of the ribs and decrease in number and malformations of the lumbar vertebrae. There were praticularly marked changes in the size, shape and capacity of the pelvis.

The other roentgenogram shows the bones of the legs in a laterolateral projection. The tibia and fibula on both sides are situated one in front of the other. They are flattened transversely and thickened in the anteroposterior diameter and curved symmetrically forward so that they are the shape of a scimitar with the convexity forward. The anterior surface of the fibula is fused with the posterior surface of the tibia along most of the length of the diaphysis. Because of this arrangement of the bones of the leg the joints are inverted, so that in place of the normal position of the malleoli the tibial malleolus is anterior and the fibular malleolus posterior. The astragalus has adapted itself to this new arrangement. The patellas are somewhat higher than their normal position and the lower ends of the femurs show a thickening of the condyles. The author has seen no similar pictures in the literature and such a condition could have occurred only in an untreated case. —*Audrey G. Morgan.*